INTRODUCTION

Peritonitis due to hollow viscous perforation continues to be one of the most common surgical emergencies to be attended by a surgeon on call duty. This may be due to persistence of the various risk factors among the general population like H.pylori infection, NSAID‘s, enteric fever and several others. This condition most of the times needs an emergency surgical intervention, a scoring system should be able to assess the need, type, and quality of the care required for a particular patient.

Realizing the need for a simple accurate scoring system in these conditions this study is undertake to estimate the use of MPI scoring system for predicting the risk about morbidity and mortality for patients with peritonitis because of hollow viscous perforation.

Several scoring systems are in place to stratify the patients with peritonitis because of hollow viscous perforation like APS, SIS, APACHE and BOEYS. Utilization of scoring systems

would be of great help in salvaging a priceless life of a patient. Our study is aimed at testing the effectiveness of MANNHEIM PERITONITIS INDEX

AIMS AND OBJECTIVES OF THE STUDY

Aim is to predict the mortality and morbidity for patients with peritonitis because of hollow viscous perforation. Assessing the outcome in these patients is helpful in choosing the modality of post op management in a particular patient.

Our study tries to assess the prognostic value of MPI scoring system in patients with peritonitis because of hollow viscous perforation, to assess this as a clinical tool in distinguish the patients based on individual operative possibility.

REVIEW OF LITERATURE

Ebers papyrus (Egypt) 1500 BC contains the first description of peritoneum. The anatomy of peritoneum and various changes in pathological conditions has been described in detail by various eminent personalities.

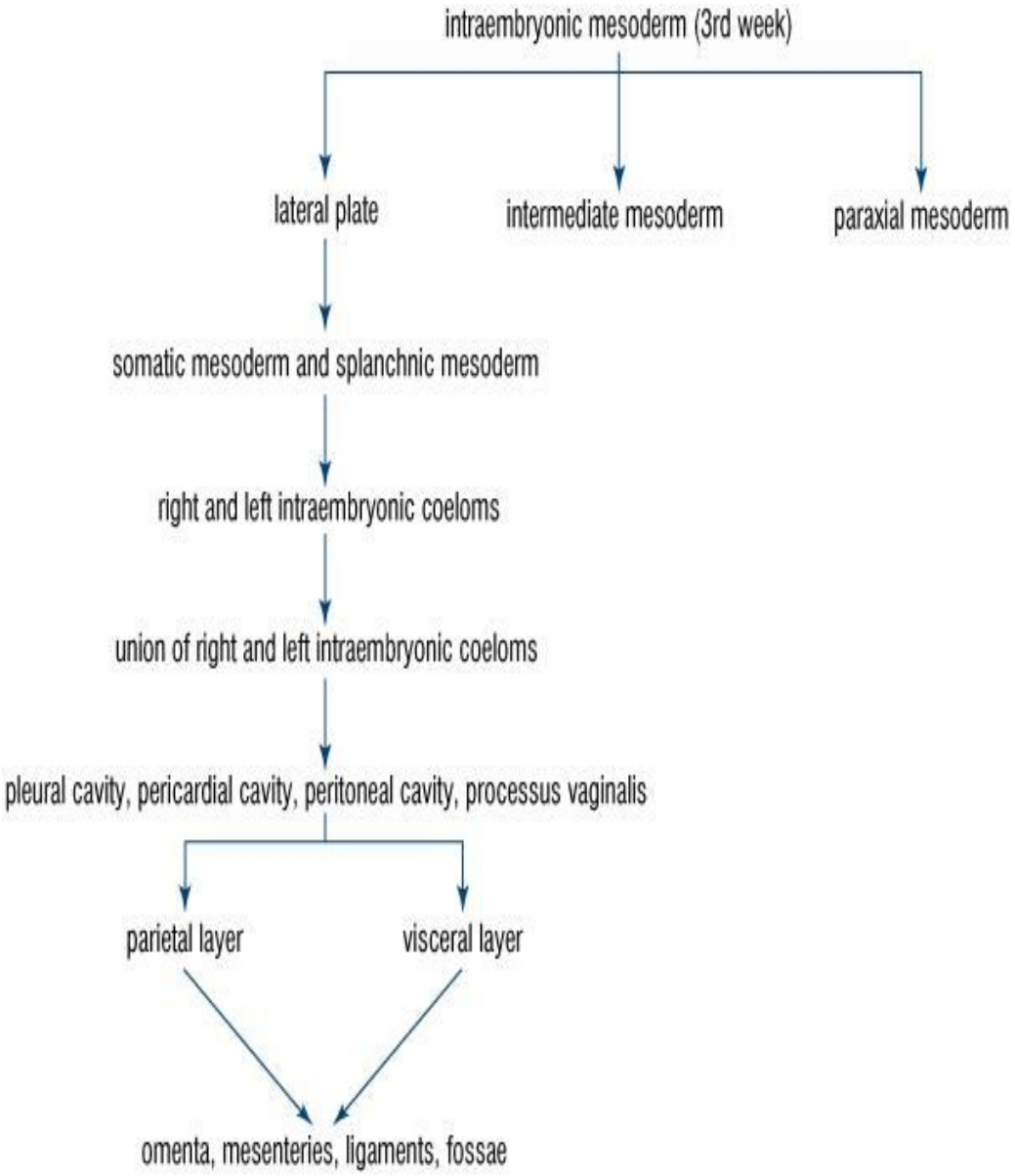
The first detailed description of the peritoneum was given by Douglas in 1730 following which numerous personalities have given their contribution to the knowledge about its structure, functions & pathological changes.

Morrison in 1894 described the right infra hepatic space (sub hepatic, hepato renal space) and vascular regenerative capacity of omentum.

Winslow in 1732 described greater and lesser omentum, lesser sac, and foramen (of Winslow)

It is an axiom that a surgeon must enter the peritoneal cavity —Prepared for anything and everything [1]

EMBRYOLOGY


intraembryonic mesoderm (3rd week)
lateral plate
intermediate mesoderm
paraxial mesoderm
somatic mesoderm and splanchnic mesoderm
right and left intraembryonic coeloms
union of right and left intraembryonic coeloms
pleural cavity, pericardial cavity, peritoneal cavity, processus vaginalis
parietal layer
visceral layer
omenta, mesenteries, ligaments, fossae

The peritoneal cavity is subdivided into compartments or spaces by 11 ligaments and mesenteries[3].

The peritoneal ligaments are duodenocolic, coronary, gastrosplenic, hepatoduodenal, gastrocolic, splenorenal, gastrohepatic, and phrenicocolic, falciform ligaments & small bowel mesentery& transverse mesocolon

These ligaments dividing abdomen as nine potential spaces:

Right subpherenic & left subphrenic space,

Right Subhepatic space,

Inframesenteric space & supramesenteric space,

Left paracolic gutter & right paracolic gutter,

Pelvic & lesser sac.

These nine potential spaces divert the fluid circulation into the peritoneum and it may useful in assessing the path of spread of infecting & malignant disease.

The left paracolic gutter is infracolic only, interrupted by phrenicocolic ligament whereas the right paracolic gutter extends into supracolic space also.

Pelvic cavity is divided into right & left by sigmoid colon & rectum, further divided in females into anterior & posterior by broad ligament, uterine tubes & uterus.

In males peritoneal cavity is a truly closed sac whereas in females the minute openings of uterine tubes provide continuity with the environment external to the body.

Extra peritoneal spaces are

Bare area of liver .

Diaphragm.

Left extra peritoneal space

Formation of adhesions & pseudo membranes may contribute to the formation of collections of abscesses in various parts of each space.

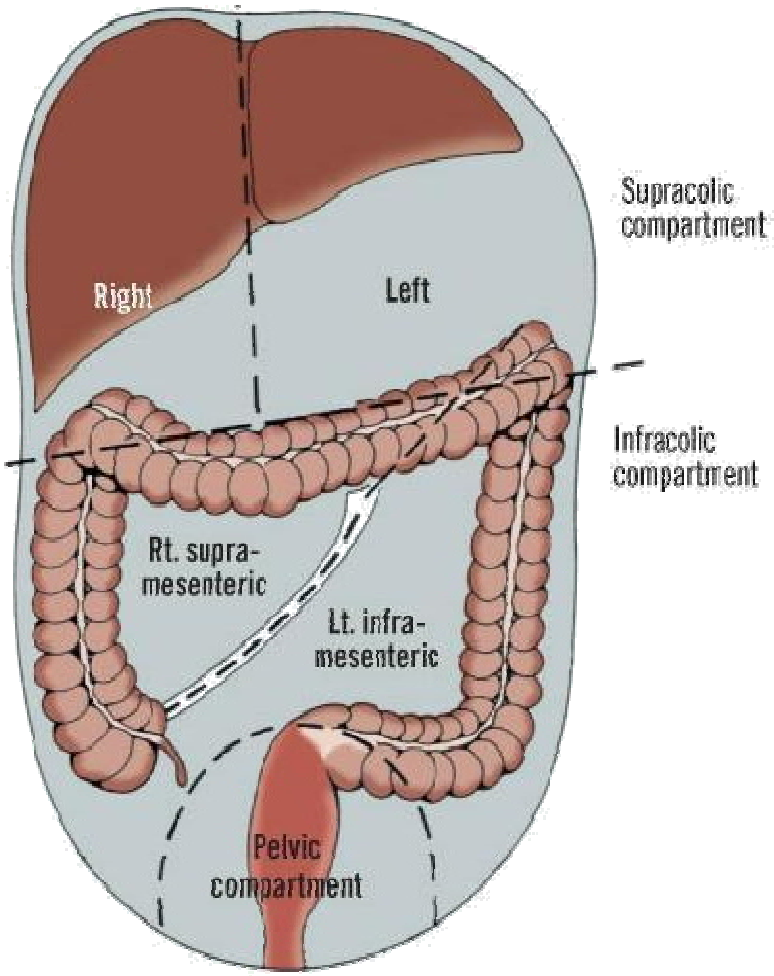
Parts of peritoneum[4]

Omenta – greater & lesser

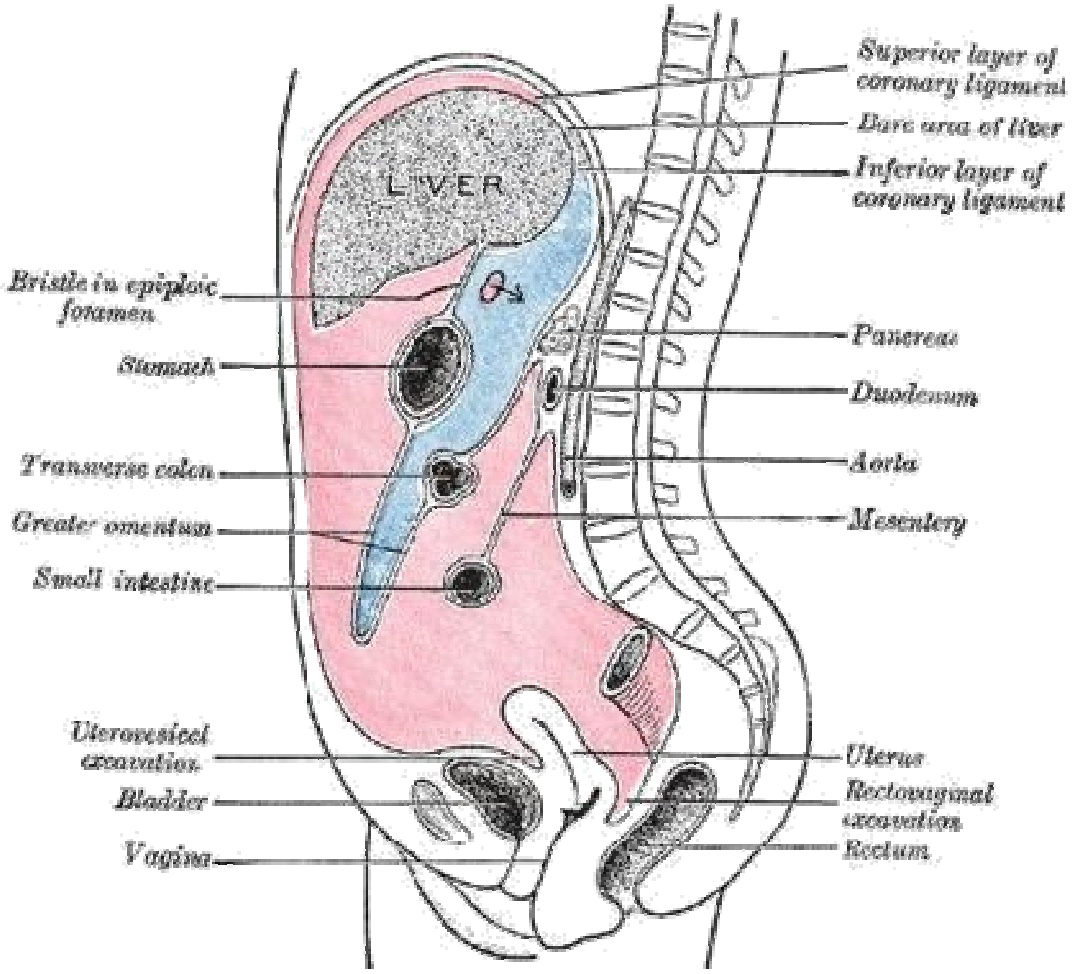
Mesentery – of small intestine, meso appendix , transverse mesocolon

Ligaments - of liver, bladder, uterus

Fossae – duodenal, ileal, intersigmoid



COMPARTMENTS OF PERITONEUM



COMPARTMENTS OF PERITONEUM – LATERAL VIEW

DRAINAGE PATTERNS

Spread of fluid in the peritoneal cavity depends upon

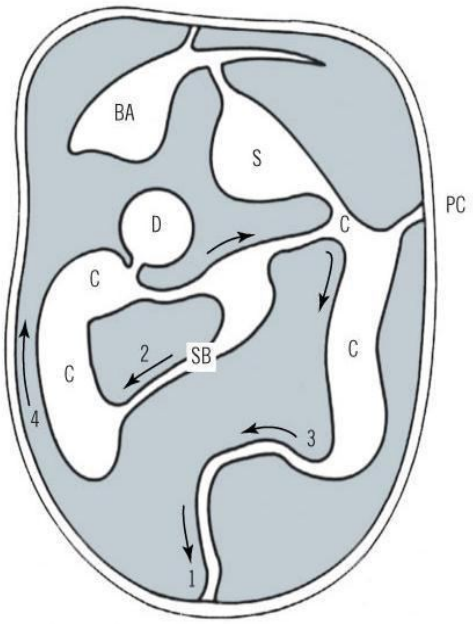
Location of source & rate of fluid production

Pressure difference in abdomen

Mesenteric partitions & peritoneal fossae

Position of the body in relation to gravity

Most common site of fluid collection is pouch of Douglas – the most dependent part of peritoneum The mesentery of small intestine & sigmoid mesocolon form barriers at which ascitic fluid may accumulate before spilling to pelvis.



Posterior abdominal wall: Mesenteric attachments and chief sites of fluid collection in order of frequency: 1, Pouch of Douglas; 2, Distal attachment of mesentery; 3, Attachment of sigmoid mesocolon; 4, Right paracolic gutter; BA, Bare area; C, Attachment of colon; D, Attachment of duodenum; PC, phrenico colic ligament; S, Attachment of stomach; SB, Attachment of mesentery of small bowel[5]

HISTOLOGY & PHYSIOLOGY[6]:

Both Parietal & visceral parts of the peritoneum have same histology – basement membrane covered by single layer of mesothelial cells. Loss of these cells produces non physiological adhesions between two parts.

Parietal layer is very loosely attached to preperitoneal fat but visceral layer is fixed firmly to subserosa of GI tract.

Peritoneal cavity is a potential space with about 50ml of isotonic fluid & less than 300 mononuclear cells.

Peritoneal fluid has water, proteins, electrolytes and diverse cellular types, provides lubrication to facilitate the movements of viscera.

With an abnormal peritoneal collection of fluids, the phenomenon of absorption is limited in lower abdomen. Absorption is more active in both subphrenic spaces, may be due to existence of specialized subphrenic peritoneum with gaps/peritoneal stomata and slit like orifice.

Peritoneum can clear bacteria within minutes conversely it may rapidly transport bacteria into systemic circulation via diaphragmatic lymhatics. Abscess formation is the final defense of this remarkable membrane if cellular, humoral and clearance defense mechanisms are overwhelmed.

Bacteria can be seen in the thoracic duct in 6 min & blood in 30 min demonstrates the clearance mechanisms of diaphragm

Functions of peritoneum[7]

Pain perception

Visceral lubrication

Fluid and particulate absorption

Inflammatory and particulate absorption

Fibrinolytic activity.

INNERVATION;

Parietal layer is innervated by somatic afferent nerves, contains many sensory fibers for sensation of pain.

Visceral layer is relatively insensitive to pain as there is no somatic afferents. Pain over the midline of abdomen.

So a perforated viscous may produce anterior abdominal wall rigidity and intra peritoneal fluid collection, produces sensation of traction or tension on the mesentery in the retroperitoneal space but not localized pain.

HOST DEFENSES OF THE PERITONEUM:

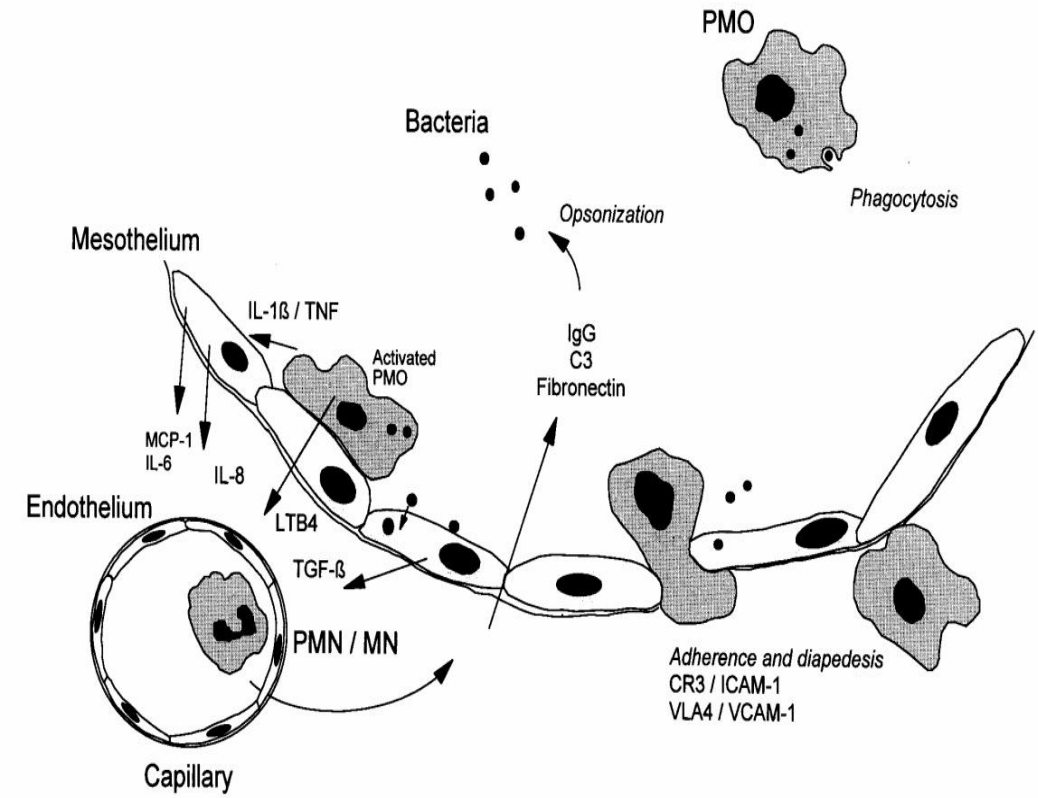


Figure 1 – Schematic representation of the effects of bacterial contamination of the peritoneal cavity on peritoneal macrophages and the involvement of the mesothelium in local host defenses.

PERITONEAL RESPONSE TO INFECTION

1. DIAPHRAGMATIC LYMPHATICS[9]

Over the diaphragm the usual smooth flat layer of cells are interrupted by a large number of inter cellular gaps called **Stomata.**(as named by VonReckling Hausen).

These act as entrance to diaphragmatic lymphatic channels called **LACUNAE.**

These lacunae are oriented parallel to muscle fibers of diaphragm and contain valves that prevent reflux of fluid back into abdomen.

They ultimately draining into substernal lymph nodes & then to thoracic duct.

FACTORS INFLUNCING UPTAKE

Mesothelial cell processes – usually in a contracted state, when relaxes - size of stomata increases.

State of diaphragm contraction –

Inspiration – diaphragm contracts – constriction of stomata

Expiration – diaphragm relaxes – opening of stomata, fluid and particulate matter gets sucked in.

3. Inflammation – increases stomata patency by inducing mesothelial cell retraction. Normally up to 30% of total lymphatic drainage of the peritoneum is by diaphragm & rest through parietal peritoneum.

CELLULAR DEFENCE

In a healthy individual the peritoneal cavity contains approximately 15 -50 mL of fluid with about 6 x 10^5 cells/ml (10).

Macrophages

Have two main functions in first-Line defense

Recognition , phagocytosis and killing

Participation in the immune response.

Opsonized microorganisms are recognized by specific receptors on the phagocyte.

The most important receptors on macrophages are the Cl (CR1) and C3receptors (CR3; recognize particle - associated C3b) and the Fc-receptors.

These recognize complexes between antigen and opsonizing antibody, e.g., IgG).

Other phagocyte receptors are fibronectin and lectin receptors8

Macrophages also seen in the submesothelial interstitium. 11

PERITONEAL FIBROBLAST

Gives signal to the intraperitoneal recruit of inflammatory bone marrow-derived cells.

Acts as sentinel cells to combine structural and immunomodulatory function. [12]

HPFBs (human peritoneal fibroblasts) provide signals stimulate the peritoneal recuite of neutrophils meantime the secretion of HPFB-derived chemokines shifts to mononuclear chemo attractants.

The peritoneal membrane contributes to inflammation by generating prostaglandins (PGI2 andPGE2) and cytokines (MCP-l, IL-I & beta;, IL-6, and IL-8) in response to cytokines released by Peritoneal macrophages (TNFa and IL-l) or directly to (parts of) bacteria.

PERITONEAL MESOTHELIAL CELLS:

Secrete plasminogen activator

Decrease in the fibrinolytic activity

Formation of fibrin adhesions

Initially helps in localizing the inflammation

EOSINOPHILS, BASOPHILS, MAST CELLS

Act by producing histamine which leads endothelial cell contraction thereby increasing the vascular permeability & influx complement factors & leukotriens.

PERITONITIS:

Peritonitis is a inflammatory response of peritoneum to irritation.

Table Classification According to Etiology[13]

I. Primary peritonitis	C. Posttraumatic peritonitis
A. Spontaneous peritonitis in children	1. Peritonitis due to blunt abdominal injury
B. Spontaneous peritonitis in adults	2. Peritonitis after penetrating abdominal Trauma
C. Peritonitis in patients with CAPD	3. Other forms
D. Tuberculous peritonitis	III. Tertiary peritonitis
E. Other forms	A. Peritonitis not by pathogens
II. Secondary peritonitis	B. Peritonitis due to fungi
A. Acute perforation peritonitis (acute suppurative peritonitis)	C. Peritonitis by low pathogenic Bacteria
1. GI tract perforation	IV. Intra-abdominal abscess
2. Bowel wall necrosis (intestinal ischemia)	A. Associated with primary peritonitis

3. Pelvic peritonitis	B. Associated with secondary peritonitis
4. Other forms	C. Associated with tertiary peritonitis
B. Postoperative peritonitis	
1. Anastomotic leak	
2. suture line leak	
3. Stump leak	
4. Other iatrogenic leaks	

PRIMARY (SPONTANEOUS) PERITONITIS

Occurring in the absence of gastrointestinal perforation is caused mainly by hematogenous spread but rarely through luminal or direct invasion of the peritoneal cavity. Impairment of the hepatic RE system and peripheral killing of bacteria by polymorphs produces bacteremia, which instantly infects peritoneal fluid, this reduce bacteria-destructing ability.

The pathogenesis of SBP unknown; however, several studies suggest bacterial translocation of bacteria within GI tract play a role .[4]

Primary peritonitis was most closely associated with cirrhosis and advanced liver disease with a low ascitic fluid protein concentration. It is also seen in patients with the nephrotic syndrome or systemic lupus erythematous, or after spleenectomy during childhood. Recurrence is common in cirrhosis and often proves fatal.

The clinical presentation simulates secondary bacterial peritonitis, with abrupt onset of fever, abdominal pain distention, and rebound tenderness. However, one-fourth of patients have minimal or no peritoneal symptoms. Most patients will have clinical and biochemical manifestations of advanced cirrhosis or nephrosis. Leukocytosis, hypoalbuminemia, and a

prolonged prothrombin time are characteristic findings. The diagnosis hinges upon examination of the ascitic fluid, which reveals a white blood cell count more than five hundred/micL and more than 25% polymorphonuclear leukocytes. A blood-ascitic fluid albumin ratio more than 1.1 g/dL, a raised blood lactate level (> 33 mg/dL), or a reduced ascitic fluid pH (< 7.31) supports the diagnosis. Bacteria are seen on Gram-stained smears in only 25% of cases. Culture of ascitic fluid inoculated immediately into blood culture media at the bedside usually reveals a single enteric organism, most commonly E coli, klebsiella, or streptococci, but Listeria monocytogenes has been reported in immunocompromised hosts.

TREATMENT

Antibiotic prophylaxis is of no proven value. Systemic antibiotics with third-generation cephalosporins (e.g., cefotaxime) or a beta-lactam-clavulanic acid combination along with supportive treatment are begun once the diagnosis has been established.

SECONDARY PERITONITIS

Secondary peritonitis due to bacterial contamination sourcing from the viscera or from other source (e.g., stab injury). It most often follows disruption of a hollow viscous.

BACERIOLOGY OF PERITONITIS

Systemic sepsis due to peritonitis occurs in varying degrees depending on the virulence of the pathogens, the bacterial load, and the duration of bacterial proliferation and synergistic interaction.

Pt for spontaneous bacterial peritonitis, peritonitis is almost invariably polymicrobial; cultures usually contain more than one aerobic and more than two anaerobic species. The microbial picture reflects the bacterial flora of the involved organ.

As long as gastric acid secretion and gastric emptying are normal, perforations of the stomach are generally sterile or associated with relatively small numbers of gram-positive organisms. Normally there are <1000 bacteria per ml in stomach secretions. No obligate anaerobes but alpha hemolytic streptococci, lactobacilli, yeasts & few oral bacteria.

Within the duodenum & jejunum there are 100 to 10000 bacteria per ml, primarily streptococci, lactobacilli, transitory oral flora & rarely enterobacter species.

Perforations or ischemic injuries of the distal small bowel (e.g., strangulated hernia) lead to infection with aerobic bacteria in about 30% of cases and anaerobic organisms in about 10% of cases. Bacterial count is 10^6 per ml to 10^7per ml. Streptococci, lactobacilli predominate, bacteroids & enterobacter are found in equal proportions.

Fecal spillage, with a bacterial load of 10^{12} or more organisms per gram, is extremely toxic. 60% of dry fecal matter is bacteria. Bacterial count is 3.8×10^{14} per mg dry stool. Streptococci, bacillus species, enterococci, E.coli, bacteroids, clostridia & anaerobic cocci form the flora.

CAUSES OF SECONDARY PERITONITIS

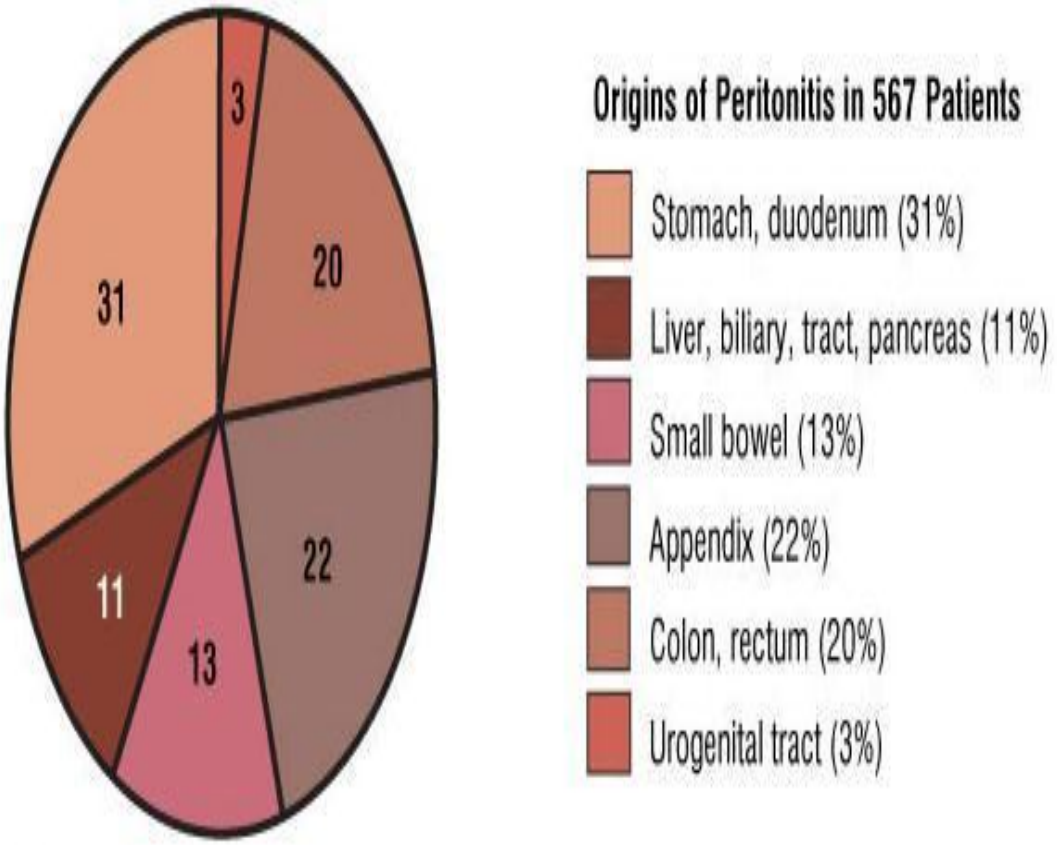
Common causes of secondary peritonitis

Esophagus – Boerhaeve syndrome, iatrogenic instrumentation, trauma, malignancy.

Stomach & duodenum – APD, analgesic abuse, malignancy, trauma.

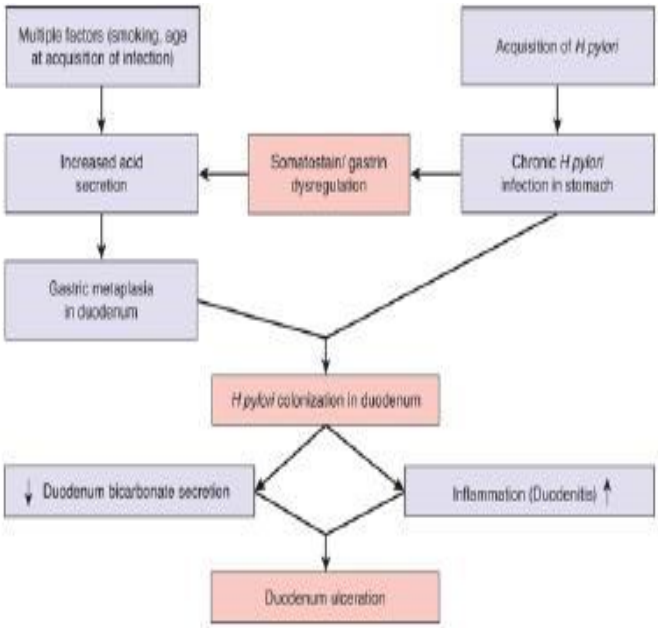
Small bowel – ischemic bowel, crohns disease, meckel`s diverticulitis, incarcerated hernia.

Large bowel - ischemic bowel, crohns disease, diverticulitis, ulcerative colitis, appendicitis



H.PYLORI & PEPTIC ULCER

H.pylori infectied patients , gastritis(antrum) were nearly 3.5 times more chance to get peptic ulcer disease than those without .70 -- 90% of patient with gastric ulcer, and about 90% of patients with duodenal ulcers, may have H.pylori infection.



PATHOGENESIS OF PEPTIC ULCER DISEASE

Sex: The ratio is 2 men to 1 woman.

Age: The highest incidence is between 45 and 55 years.

Most often a peptic ulcer that perforates is situated on the anterior surface of the duodenum; much less frequently it is situated on the anterior surface of the stomach, usually near the lesser curvature or the pyloric antrum.

Rarely an ulcer on the posterior wall of the stomach perforates into the lesser sac.

In 80 per cent of cases, there is a history — often a long history —of peptic ulceration.

In 20 per cent there is no such history.

It is a silent chronic ulcer that perforates, especially in those patients who are being treated with cortisone[19].

NSAID induced GI damage:

Risk for developing GI complication are 3 times high in those consuming than those not.

This may goes upto five times age more than 60. In elder population, need surgery related complication is 10 times, and the risk that they will death due to a GI etiology was 5 times more[20].

>20% of patients with perforation of >60 years age group have had history of NSAID consumption

NSAIDs cause GI damage by Topical biochemical reactions – inhibition of COX prevents mucosal repair because of decreased prostaglandin synthesis leading to decreased mucosal blood flow.

Inflammatory response due to more permeable that favours luminal factors access to GI mucosa – NSAIDs are uncouplers of oxidative phosphorylation in mitochondria with consequent decrease in intracellular ATPs loss of cytoskeletal control over tight junctions.

Smoking, Stress, and Other Factors

Previous studies reported that people smoking are 2times increased risk to peptic ulcer disease as those not. Smoking increases acidity and GI reflux. It also diminish gastro duodenal PG secretion and HCO3 production by pancreas.

Curling descries DU/ duodenitis in burn patients, later, Cushing describes the presence of APD in patients with head injury (Cushing ulcer). Patients with ulcer related complications like bleeding, perforation, obstruction are promoted due to stress.

Now, the usage of **cocaine** is associated with juxta pyloric peptic benign ulcers and high chance to perforate.

Alcohol was also a risk factor, no recent data confirm it.

PERFORATION IN ENETRIC FEVER

- Enteric fever is caused by salmonella typhi
- Most common in 21-30 age group.
- Disease is common in lower socioeconomic status mainly due to contaminated water supply.
- Majority of the perforations occur within 2 wks of illness.
- Mechanism – hyperplasia & necrosis of Peyers patches in terminal ileum leading to tissue damage.
- Necrosis may end up with full thickness perforation.

Treatment: Closure of perforation & appropriate antibiotics.

BILIARY PERITONITIS

- Causes are
- Perforated cholecystitis
- Post cholecystectomy
- Cystic duct stump leakage
- Division of accessory duct
- Bile duct injury
- T - tube drain dislodgement
- Following other operations/ procedures
- Leaking duodenal stump post gastrectomy
- Leaking biliary enteric anastomosis
- Leaking around percutaneous placed biliary drains.

Unless the bile has extravasated slowly and the collection gets shut off from the general peritoneal cavity usually the bile leak leads to diffuse peritonitis due to the high irritant nature of the bile, the patient is often jaundiced due to absorption of peritoneal bile.

Treatment -Laparotomy or laparoscopy done to evacuate the bile, peritoneal lavage and source of bile leakage identified and corrected.

MECONIUM PERITONITIS

Meconium normally sterile, it is a mix of epithelium, mucin, salts, lipids and bile.

Meconium peritonitis was an aseptic peritonitis with meconium entering the peritoneum via an perforated intestine which may be because of GI obstruction.

It is considered when a baby is born with a tense abdomen, is vomiting and in whom there is failure to pass meconium.

Radiography shows free air in the peritoneal cavity, fluid levels, fluid in the abdomen and calcification most often on the surface of liver or spleen.

Treatment;

Laparatomy - closure of perforation and drainage of peritoneal cavity.

BACKGROUND FOR SCORING SYSTEMS

Many scoring system were developed for assessing mortality and morbidity due to peritonitis.

These scoring systems that help to asses the severity of the intra-abdominal infection are essential to:

1. The effectiveness of different treatment regimens,

2, To utilize ICU care

Help to identify individual risk to select them who may need a aggressive operative management

3.To inform patient´s attenders with higher objectivity

Existing scoring systems

	Scores predicting mortality	Scores predicting morbidity
Scores not requiring operative information	ASA APACHE-II Sickness Assessment Boey Score Hacetteppe Score Physiological POSSUM	APACHE-II Veltkamp Score VA Pneumonia Prediction Index VA Respiratory Failure Score
Scores requiring operative information	Mannheim Peritonitis Index Reiss Index Fitness Score POSSUM, P-POSSUM Cleveland Colorectal Model Surgical Risk Scale	POSSUM, P-POSSUM

ASA SCORING

Inspite of subject variation and observer variation of measuring, its used for several years, remain the score mostly useful in emergency. ASA SCORING was not developed for predict mortality but it shown a best estimate the risk of mortality ,easy to score. This is subjective, may be applied by various anesthetists. This scores are inter observer variation, suggests that it is really an expert risk assessment .

Table 2b: Summary of the 6 studies observing mortality after emergency surgery in the elderly

	ASA I	ASA II	ASA III	ASA IV	ASA V
Total deaths	31		62	143	41
Total cases	511		338	329	46
Mortality	6%		18%	44%	89%

BOEY SCORING SYSTEM

1. Presence of Shock at the time of admission (systolic BP<90 mmHg),
2. Severe co morbid medical problems (ASA III–V), and
3. Late presentation (symptoms >24 h).

Risk Factors	No. of Risk Factors	Risk of Mortality Boey
	0	0
Preoperative BP < 100 mmHg	1	10%
Delayed presentation > 24 h	2	45.5%
Major medical illness present	3	100%

ADVANTAGES

Simple , easy to remember and apply.

DISADVANTAGES

Does not consider various other physiological factors which do have a significant role in predicting the patients condition. Less accurate .

HACETTEPPE SCORE

– used in peptic ulcer perforation

The four variables in the study

The associated of a severe pre existing medical problems,

ARF,

WBC > 20×10^9/l,

MALE.

SICKNESS ASSESMENT

Kennedy *et al* - first described this scoring system.

1. Hypotension

2. chronic illness

3. whether independent or not ,self-care.

This parameters are clearly explained. For the group with a Sickness assessment score of 0, there is no death. Mortality in group of 1,2,3 parameters presence is 52%, 60% , 100% .

Not widely used.

FITNESS SCORE

Playforth *et al* in 1987 introduce it and used 26 factors weighted arbitrarily from 1 – 4.

Difficulty of scoring 26 factors preoperatively, some are perforation and obstruction , malignancy are not present preoperatively

REISS INDEX

Factors considered :

1.patients Age

2.Urgency of surgery

3.ASA

4. malignancy

5.Pre op Diagnosis

At emergency surgery where the diagnosis is not known this score not useful, meanwhile lower to the ASA score in predicting morbidity &mortality. Scoring by Reiss Index / Fitness Score can be useful in this situation.

SEPSIS SCORE

These are

1.APACHE

2. SAPS

3.Sepsis score

4. Multiorgan failure score

5.MPI

Scores for morbidity

1. Veltkamp score – they are eleven pt, disease, surgery related factors are taken .Minor complications are less-successfully predicted hence less commonly used.

2. **VA respiratory failure prediction index**

Score weightage were give to nature of surgery, emergency (< 12 hours after admission), albumin, urea, co-morbid status, RS System function, history ,age. The score > 40 predicts chance of respiratory system failure --31%.

APACHE II:

Now , the most accepted score is APACHE II, which taken various factors during the 1st 24 hour in the ICU with age and previous health status of the patients.

The originally formed to judge the mortality risk. It was primarily designed to stratify ICU admissions.

DRAWBACKS

The primary intra op findings are not considered in the data collected. These data have a significant bearing on the outcome of the patient. These are especially important if the APACHE was calculated after the primary surgery. The primary surgery would alter the physiological variables used to calculate the APACHE score.

Mortality prediction is less accurate

It is difficult to use this scoring system for postoperative monitoring because of the need to measure numerous laboratory parameters when deciding therapies for patients with unsuccessful initial treatment

The acquisition of so many factors shall make it less quickly measured compare to other scoring systems.

POSSUM SCORE

PHYSIOLOGICAL AND OPERATIVE SEVERITY SCORE for CALCULATE of mortality based on Copeland, Jones and Walters Br J Surg(1991)

Scores calculated taking into consideration 2 parameters

Physiological severity

Age, CVS signs , systolicBp, RS signs, pulse , GCS ,HB, total count, urea, K,Na& ECG

Operative severity

Number of procedures, blood loss, peritoneal soiling, cancerous nature , operative severity & type of procedure .

Considered to be midway between too simple ASA scoring and too complex APACHEII scoring system.

Uses 12 physiological variants and 6 operative variants Drawbacks

Tends to overestimate the mortality in low risk patients

Tends to overestimate if used in other specialties.

P POSSUM –Portsmouth predictor equation for mortality –

Prytherch et al Br J Surg 1998 introduced the corrected version of the scoring system.

This scoring is more accurate than the original POSSUM scoring but it still overestimates the mortality in low risk patients. Higher the risk more is the accuracy of the scoring system. There have been new versions of this scoring system like V-POSSUM used specifically for specialties.

Predicted death rate=$1/ (1+ e^{-R})$

Where R is (0.1692×physiological score) + (0.1550×oerative score) -9.065 in POSSSUM R = (0.13×physiogical score) + (0.16 ×operative score)-7.04 in P-POSSUM

THE MANNHEIM PERITONITIS INDEX

Mannheim Peritonitis Index (MPI) was developed by Wacha and Linder in 1983.

It was developed based on the retrospective analysis of data from 1253 patients with peritonitis, in which 20 possible risk factors were considered. Of these only 8 proved to be of prognostic relevance and were entered into the MPI, classified according to predictive powers.

Since then Mannheim Peritonitis Index has been used because of its accurateness and simplicity to predict the morbidity and mortality in patients with hollow viscous perforation.

THE MANNHEIM PERITONITIS INDEX

RISK FACTOR	SCORES
Age > 50 years	5
Female sex	5
Organ failure	7
Malignancy	4
peritonitis duration > 24 h	4
Non –colonic Origin of sepsis	4
Diffuse generalized peritonitis	6
Type of Exudate	
1.Clear	0
2.Cloud, pus	6
3.Fecalunt	12

Kidney failure means serum creatinine > 177 umol/L / urea > 167mmol/L or oliguria 20ml/hr; Pulmonary insufficiency means PO2 < 50 mmHg / PCO2 > 50 mmHg;

Intestinal obstruction or paralysis > 24hours / complete mechanical ileus,

Shock means

systolic blood pressure<90mm of hg,

MAP<60mm of hg

CLINICAL FEATURES

Localized peritonitis

The clinical course is determined by the manner in which adhesions form around the affected organ.

Inflamed peritoneum loses its glistening surface and becomes reddened & velvety. Flakes of fibrin appear and causes loops of intestines to become adherent to one another and to the parietal wall.

There is an outpouring of serous inflammatory exudates rich in leukocytes and plasma proteins that soon become turbid; if localization occurs, the turbid fluid becomes frank pus.

The greater omentum, by enveloping and becoming adherent to inflamed structures, often forms a substantial barrier to the spread of infection.

DIFFUSE PERITONITIS

A number of factors may favour the development of diffuse peritonitis

Speed of peritoneal contamination – is the prime factor. Any hollow viscous if perforates before localization has taken place; there is a gush of contents into the peritoneal cavity which may spread to a large area almost instantaneously.

Stimulation of peristalsis – hinders localization.

Virulence of the organism.

Young children have a small omentum which is less effective in localizing the infection.

- Disruption of localized collections – due to injudicious and rough handling.
- Deficient natural resistance.

Clinical features;

- Abdominal pain worse on movement
- Guarding/ rigidity of abdominal wall
- Pain/ tenderness on rectal/ vaginal examination
- Pyrexia
- Raised pulse rate
- Absent or reduced bowel sounds
- SIRS in later stages

INVESTIGATIONS:

Raised white cell counts

Erect chest x ray or lateral decubitus - air under diaphragm or beneath the abdominal wall

A radiograph of the abdomen – may show dilated gas filled loops of bowel (consistent with paralytic ileus).

USG and CT scanning – may be helpful in some patients by identifying the cause of peritonitis

Peritoneal aspiration – usually done under USG guidance. Bile stained aspirate indicates perforated peptic ulcer or gall bladder, purulent aspirate indicates bacterial peritonitis.

Treatment

Fluid and electrolyte replacement, operative control of sepsis, and systemic antibiotics are the mainstays of treatment of peritonitis.

A. General Care:[23]

IV fluids - The infusion of fluid into the peritoneum must be replaced by an appropriate amount of intravenous fluid. If systemic toxicity is evident or if the patient is old or in fragile health, a central venous pressure (or pulmonary artery wedge pressure) line folley"s catheter is inserted; a I/O chart is kept

Care for septicemia –Cardiovascular suppotive agents & mechanical ventilation in an intensive care unit are used in patients with advanced septicemia.

Antibiotics - Loading dose of iv antibiotics directed against the anticipated bacterial pathogens is given after culture.

Empirical antibiotics started are third-gn cephalosporin, amikacin, piperacilin + tazobactam, imipenem + cilastatin / azetreonam for gram-negative coliforms, clindamycin or metronidazole to cover anaerobic organism.

B. Operative Management:

Control of sepsis - The objectives of labrotomy for peritonitis are to clear all infected contents, treat the cause, and control future complication.

A midline incision offers the best surgical exposure. contents for cutures of fluid ,tissue were taken once peritoneum opened.

Occult pockets of infection are located by thorough exploration, and contaminated or necrotic material is removed.

The primary disease is then treated. This may require resection (e.g., ruptured appendix or gallbladder), repair (e.g., perforated ulcer).

PERITONEAL LAVAGE

For peritonitis, lavage with large amount (> 3 L) of warm isotonic crystalloid solution clears all matter as well as blood clots and fibrin.

Bassett[23] in his book on exploration of the abdomen and the maneuvers necessary to carry it out suggested three sequences that may be used: regional, systemic, and circular.

The following sequence is drawn from Bassett's regional route:

Inspect the abdomen.

Note any obvious pathology that

(a) may need immediate treatment (i.e., a ruptured spleen) or

(b) may contraindicate further exploration (i.e., a perforated colonic diverticular abscess).

- If the clinical status of the patient warrants further exploration,
- Examine the transverse colon. Pull the colon downward.
- Examine the supracolic region from right to left.
- Pull the transverse colon upward.
- Examine the infracolic region from right to left.
- Examine the pelvic cavity.
- Do not forget to examine the greater omentum.

Torsion of the omentum may be the cause for pain abdomen.[24] Idiopathic infarction, especially at the right lower end, is obvious.[25]

If this or any other system is used habitually, it will become automatic.

As Bassett[23] said: "**The goal is to achieve gentleness, accuracy, thoroughness, and speed."**

C. POST OPERATIVE CARE

Intensive surgical care monitoring& ventilatory support is needed if the patient is unstable & frail.

Obtaining hemodynamic stability to perfuse all vital organs is the immediate objective, and this may entail the use of cardiac inotropic agents besides fluid and blood product supportive measures.

Antibiotics were to be give for 7-10 days, based on the nature of organism.

Clinical response is monitored by,

- Well-maintained perfusion and maintained output,
- Absence of fever & decrease of wbc count,
- Resolution of ileus, and
- A returning well-being sense.

The improvement varies based on duration & severity of peritonitis.

The early removal of all unnecessary catheters (central venous ,NG tube,arterial. urinary,) is to be ensured.

Drains are removed or advanced once drainage decreases & when its serous in type.

COMPLICATIONS:

With modern treatment diffuse peritonitis carries a mortality < 10%.

SYSTEMIC COMPLICATIONS OF PERITONITIS

- Bacteremia / endotoxic shock
- Bronchopneumonia / respiratory failure
- Renal failure
- Bone marrow suppression
- Multi system failure

INTRAABDOMINAL COMPLICATIONS

- Adhesive small bowel obstruction
- Paralytic ileus
- Residual abscess and recurrent abscess
- Portal pyemia
- Liver abscess.

SURGICAL SITE INFECTIONS

Clinical features of an abdominal/ pelvic abscess:

- Malaise
- Sweats with or without rigors
- Abdominal/ pelvic pain
- Anorexia and weight loss
- Swinging pyrexia
- Symptoms from local irritation: hiccough (subphrenic), diarrhea & mucus (pelvic).
- Localized abdominal tenderness.

The primary cause of prolonged hospital stay after the operation in patients with secondary bacterial peritonitis is pulmonary and MODS & this is despite the technical success of the initial operation. Peritonitis may induce a systemic inflammatory response in which the lungs succumb more often and earlier than other remote organs.[26]

Although pulmonary failure alone did not predispose to death, ARDS and MODS are both associated with increased mortality.

A primary mediator of remote organ injury after peritonitis is the neutrophil, although such injury is multifactorial and complex in nature.

MATERIALS AND METHODS

SOURCE OF DATA

Total of 50 patients admitted with peritonitis due to hollow viscous perforation who came to GOVT STANLEY MEDICAL COLLEGE &HOSPITAL,CHENNAI From January 2014 to august 2014.

METHODS

Diagnosis of peritonitis due to hollow viscous perforation made by History and clinical examination

X-ray chest PA view with both domes of diaphragm which shows air under diaphragm.

Detailed history of presenting illness and history suggestive of chronic health disorders such as cardiac, renal, hepatic conditions noted.

All biochemical investigations done on admission and relevant clinical details noted.

Standard operative procedures was followed for different causes of perforative peritonitis

Mortality means as patient died during the admission period.

Morbidity assessed in terms of post operative complications such as

- Pneumonia
- lung atelectasis,
- Wound infection,
- Acute myocardial infarction or heart failure,
- Intra abdominal collection,
- Acute renal failure and urinary tract infection.

INCLUSION CRITERIA

Patients with clinical suspicion and investigatory support for the diagnosis of peritonitis due to hollow viscous perforation who are later confirmed by intra op findings.

EXCLUSION CRITERIA

Patients with hollow viscous perforation due to trauma

Patients with any other significant illness which is likely to affect the outcome more than the disease in study.

Once diagnosis of peritonitis had been determined by operative findings, the patient was enrolled into the study. Using history, clinical examination and lab values risk factors found in MPI were classified according to values indicated and individual variable scores were added to establish MPI score. The cases were first grouped into three, as described by Billing: those below 21 pts, between 21-29 pts, and those above 29 pts.

In addition to personal data such as name, age, sex, etc., the following information was registered: file number; dates of admission and discharge from the hospital; days hospitalized; date of surgery and information related to illness (surgical findings, medical treatment and evolution of illness).

Patient evolution was followed, occurrence of complications and discharge due to improvement or death. Time elapsed from initial diagnosis to moment of event (death or discharge from hospital) was determined.

Out-patient follow-up was continued for 30 days to establish perioperative morbidity and mortality. The minimum possible score was zero, if no adverse factor were present, and maximum was 47 if presence of all were confirmed. Analysis was done with each variable in the scoring system as an independent predictor of morbidity or mortality and the scoring system as a whole.

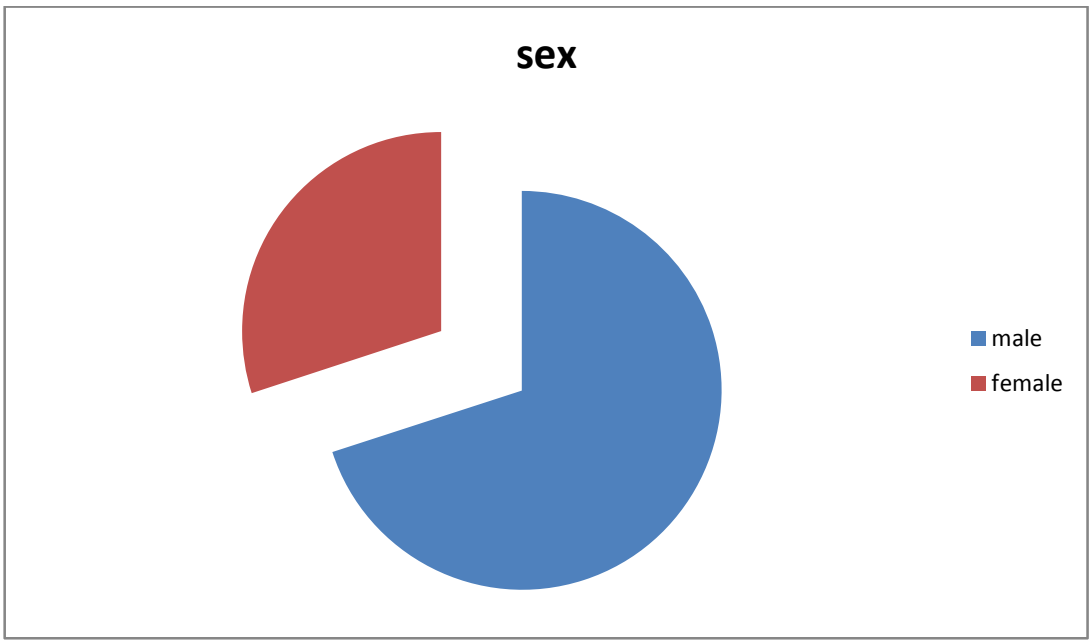
STATISTICAL ANALYSIS

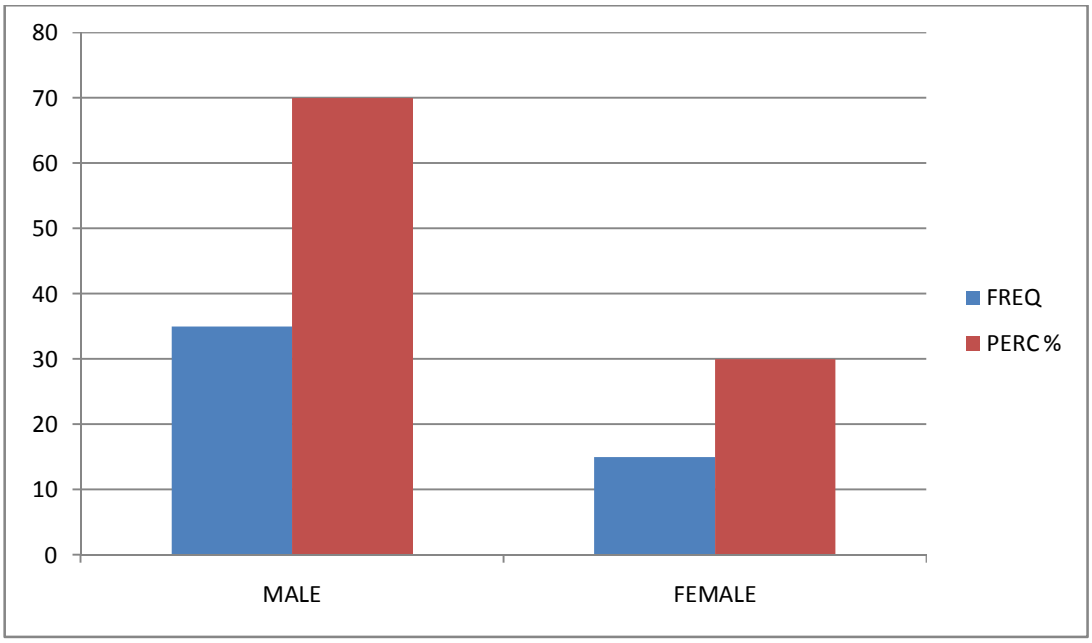
The data was analyzed using SPSS software version 16.3.Each variable in the MPI score along with other patient variables was analyzed using chi square analysis with various outcomes that were noted in the study. P value <0.05 was taken as significant in this study.

The results were averaged (mean + standard deviation) for each parameter for continuous data and numbers and percentage for categorical data presented in table and figure. Proportions were compared using Chi-square test of significance.

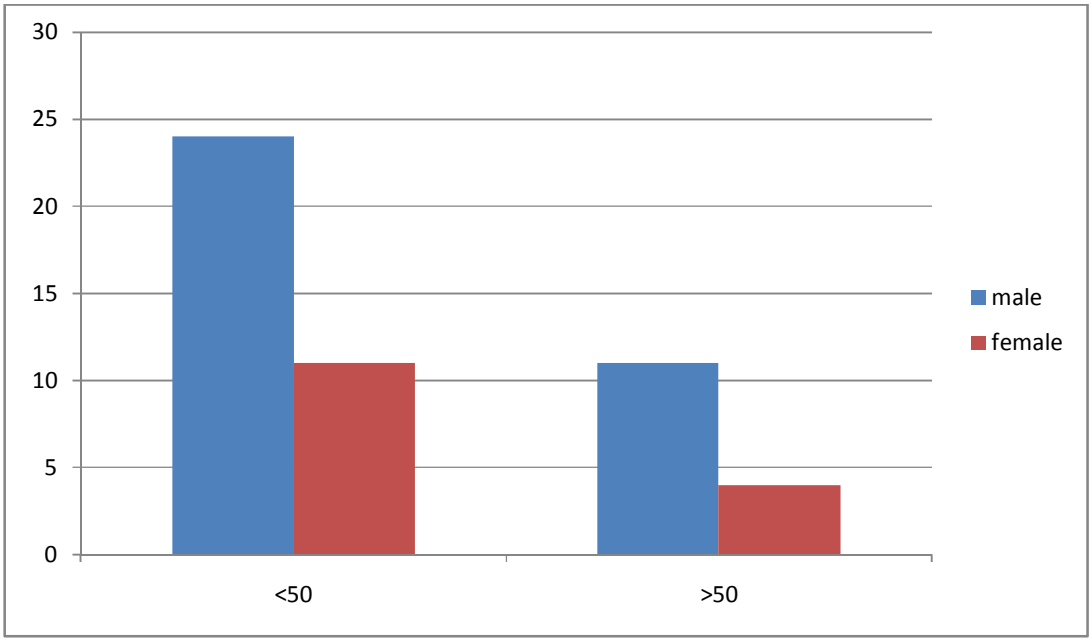
RESULTS

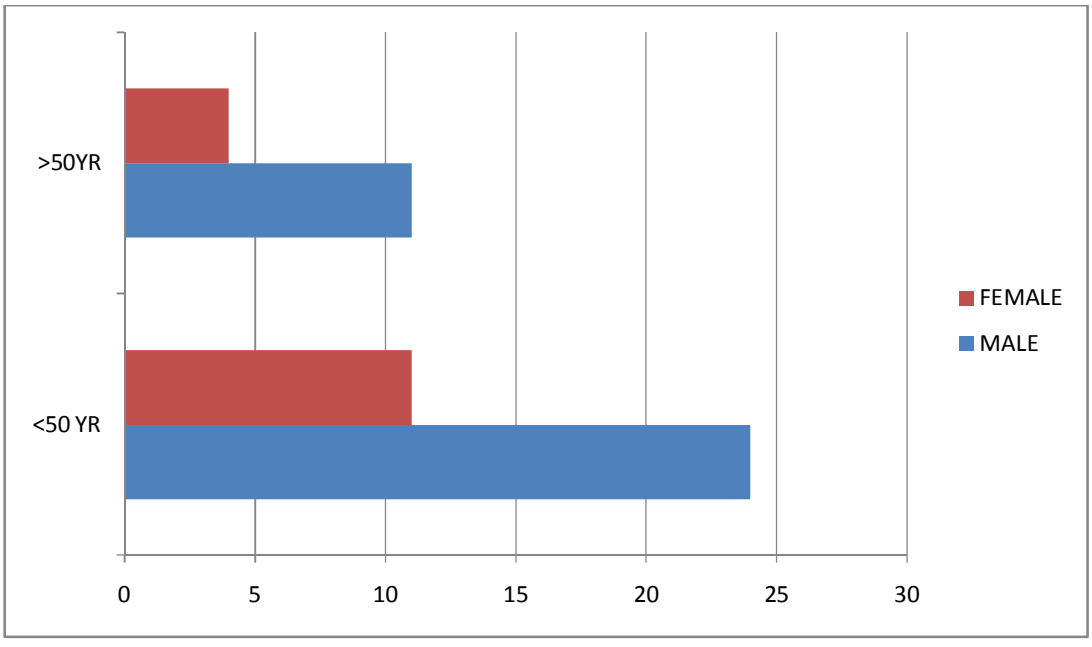
SEX DISTRIBUTION





AGEWISE DISTRIBUTION





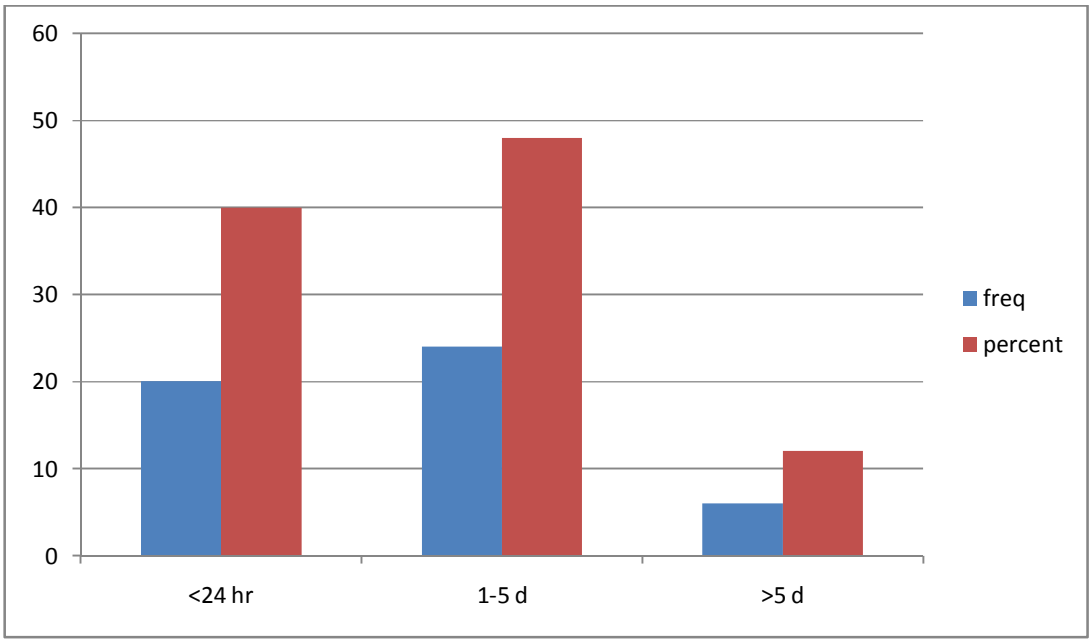
	FREQUNCY	PERCENTAGE(%)
MALE	35	70%
FEMALE	15	30%
TOTAL	50	100%

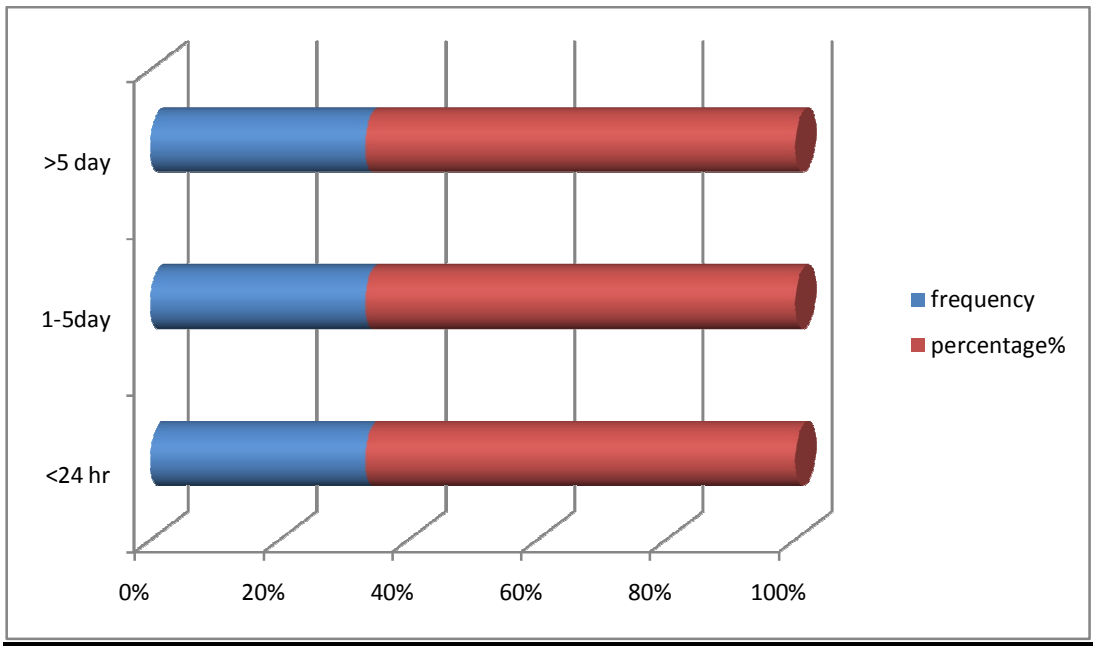
	MALE	FEMALE	FREQUENCY	PERCENTAGE %
<50	24 68.57 %	11 31.43%	35	70
>50	11 73.33	4 26.67	15	30
TOTAL	35 70%	15 30%	50	100

Males accounted for 70% of the patients in the present study whereas most of the other studies show equal sex distribution.[27, 28] 70%% of the patients were less than 50 years of age which is nearly similar to earlier studies.

DURATION OF PAIN

Duration of symp	frequency	Percentage %
<24 hr	20	40
1-5 days	24	48
>5 days	6	12
Total	50	100


60
50
40
30
20
10
0
<24 hr
1-5 d
>5 d
freq
percent


>5 day
1-5day
<24 hr
0%
20%
40%
60%
80%
100%
frequency
percentage%

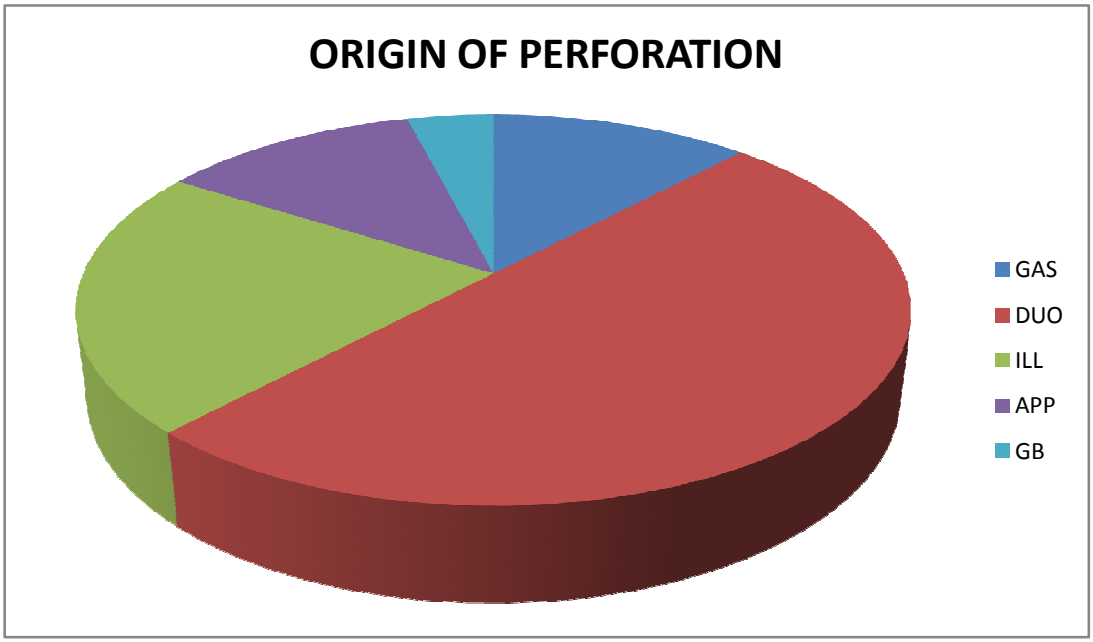
DURATION OF PAIN

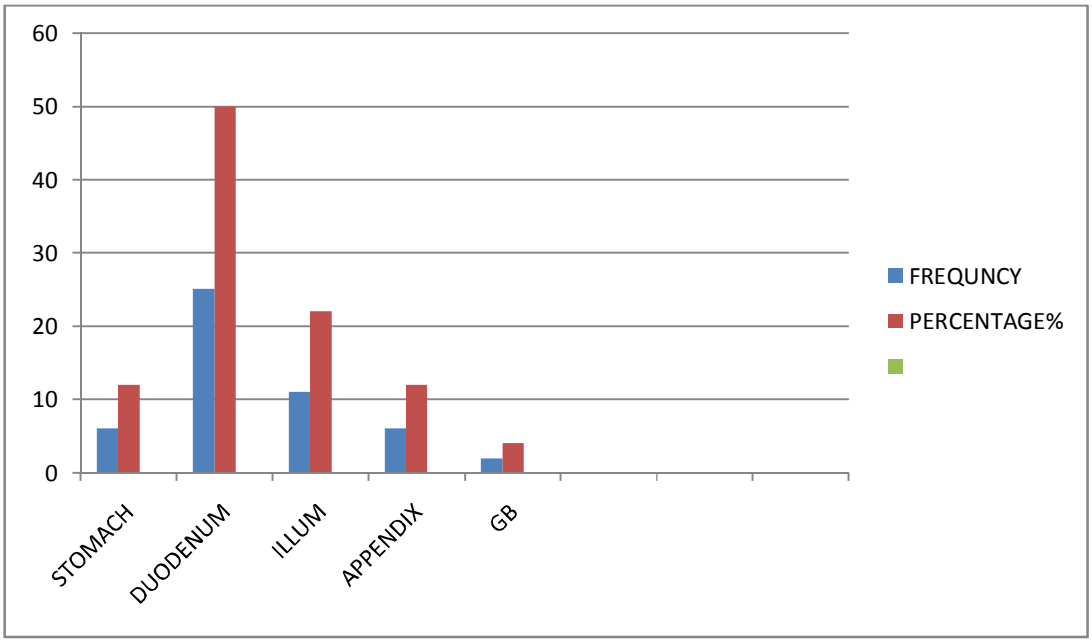
About 40% of the study group presented with pain abdomen of less than 24 hours duration.

It was seen in this study that longer the duration of pain higher was the morbidity in the post op period.

SITE OF PERFORATION-STATISTICS

ORIGIN	FREQUNCY	PERCENTAGE %
GASTRIC	6	12
DUODENUM	25	50
ILEUM	11	22
APPENDIX	6	12
GB	2	4

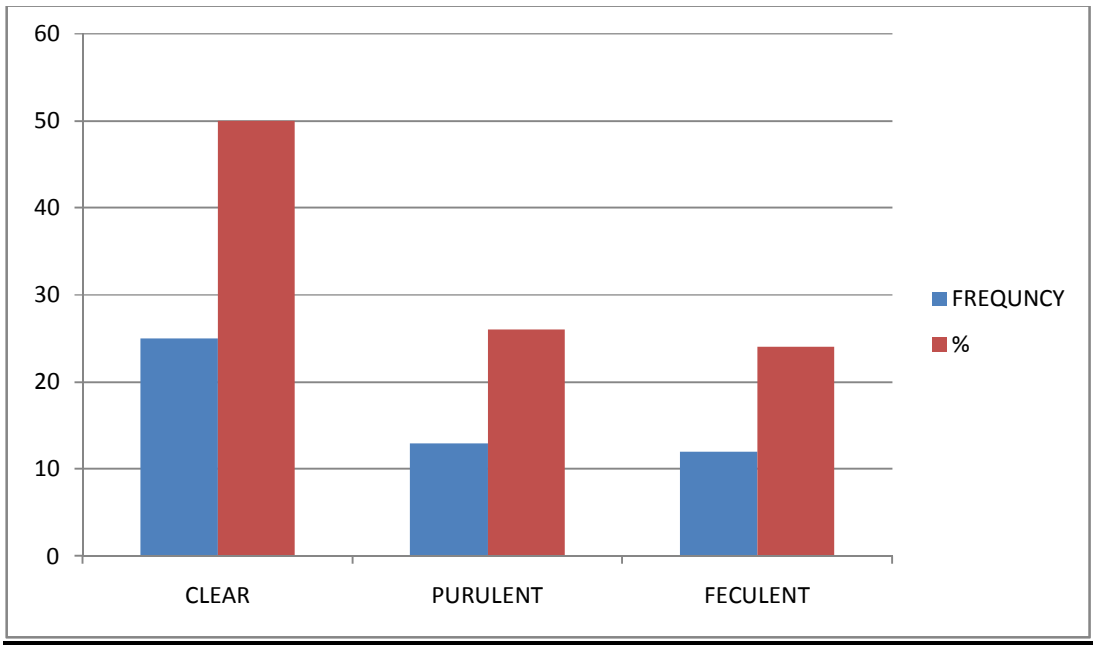


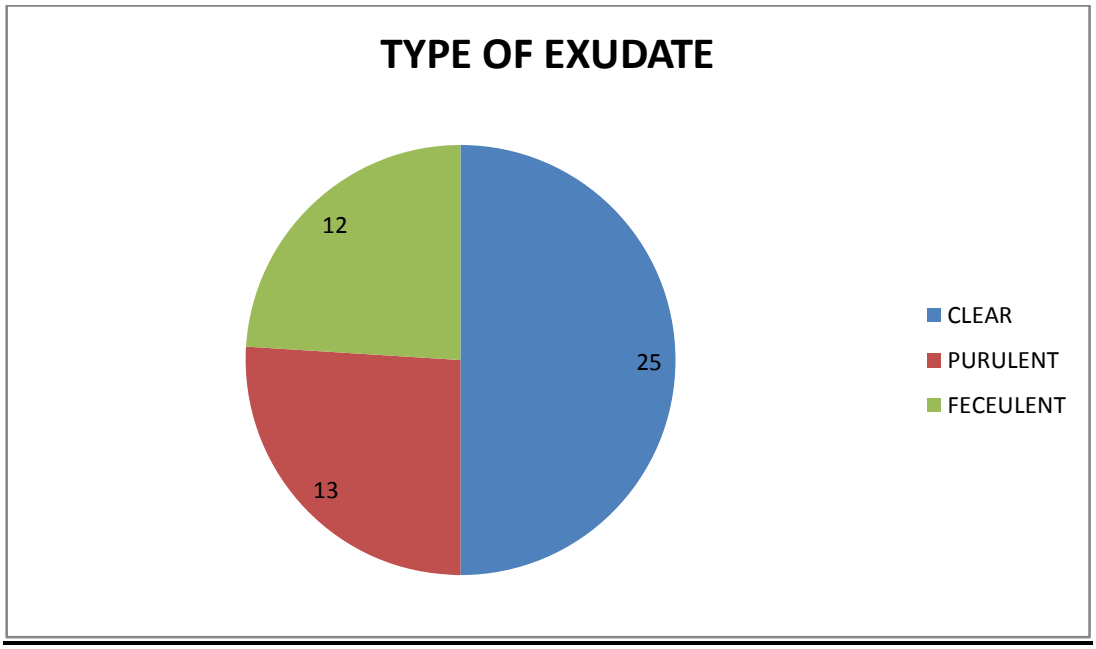


The most common site of perforation was duodenum(50%),ileal perforation being the next common .

EXUDATE-STATISTICS

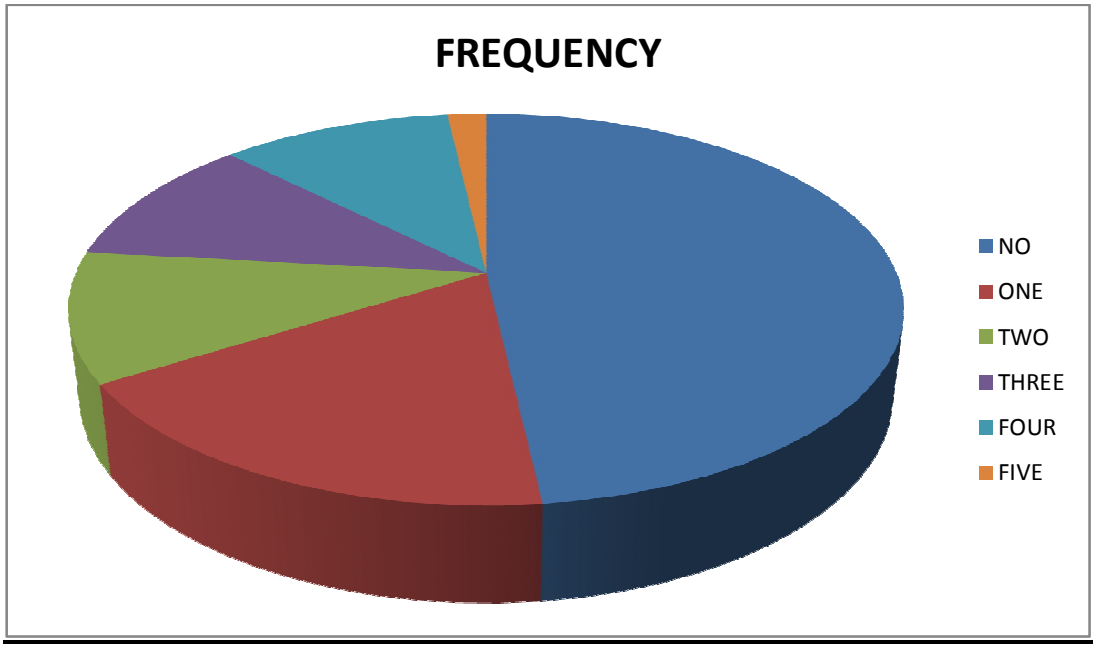
TYPE	FREQUENCY	PERCENTAGE %
CLEAR	**25**	**50**
PURULENT	13	26
FECULENT	12	24
TOTAL	50	100

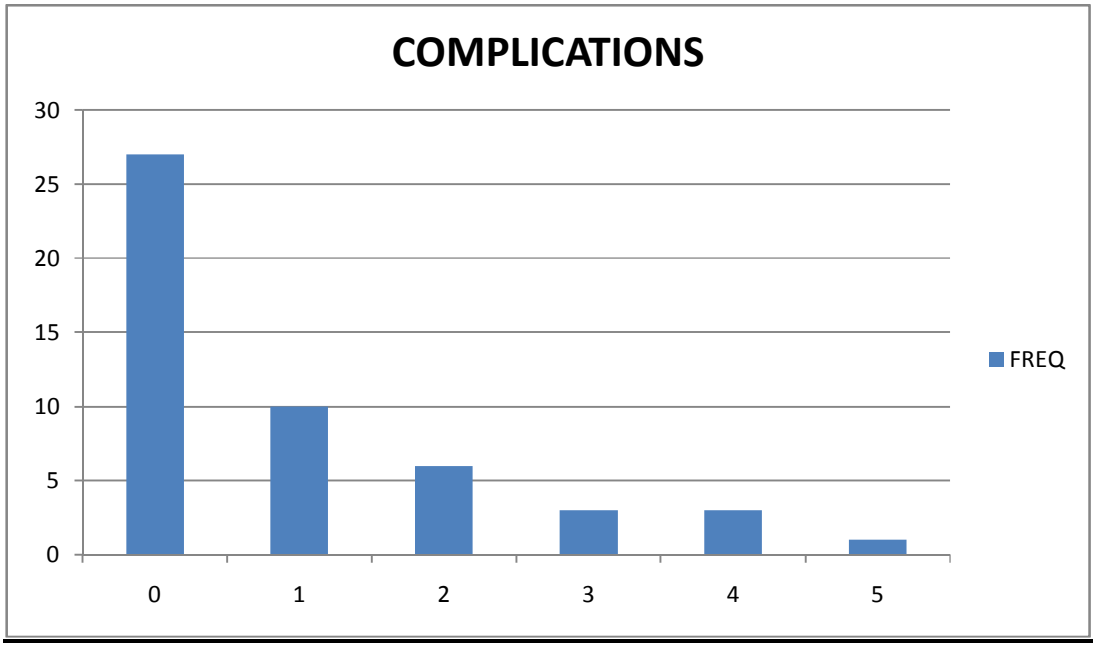




Only 12% had feculent collection noted intra op, majority (50%)had clear fluid.

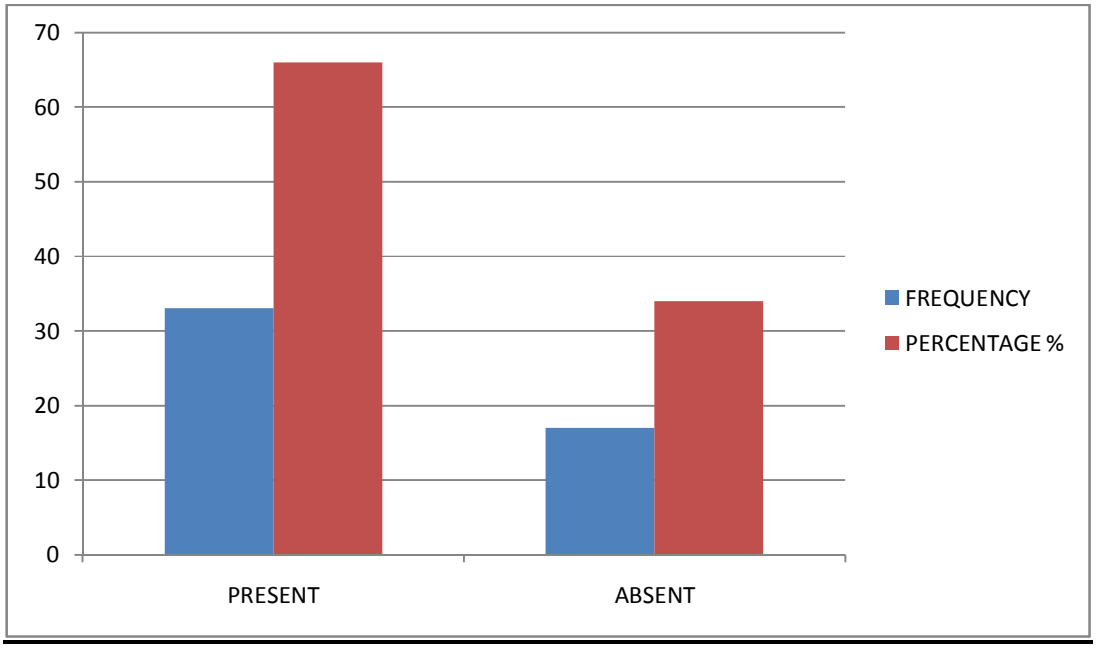
COMPLICATIONS- STATISTICS

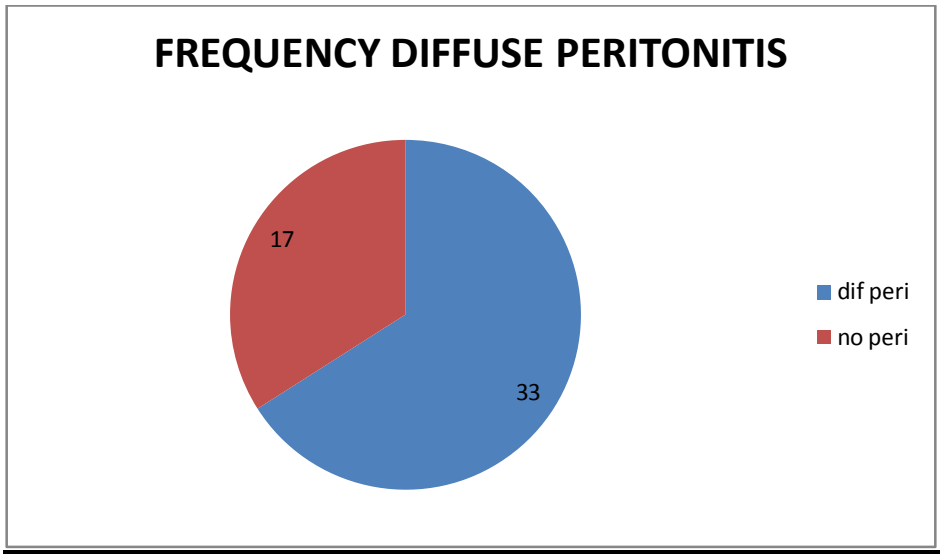




Here majority (>50%) of patients don't develop any complication.

DIFFUSE PERITONITIES





66% of the study population presented with diffuse peritonitis. Only two case(4%) was perforation due to gall bladder pathology.

About 34% of study population doesn't develop any peritonitis,

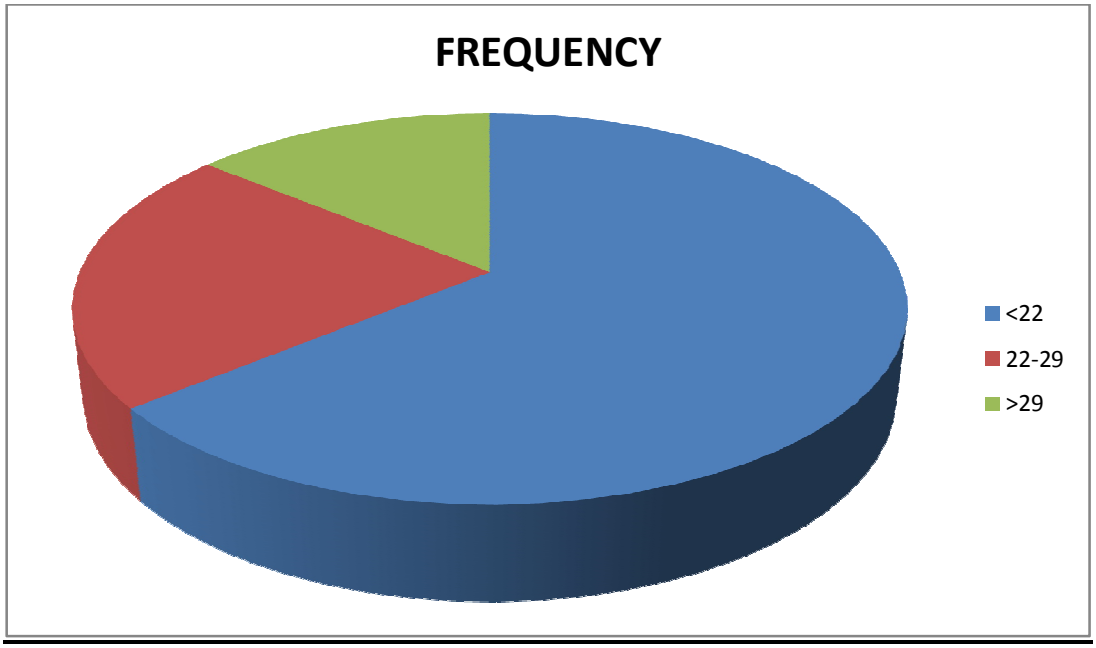
1. Site of perforation,

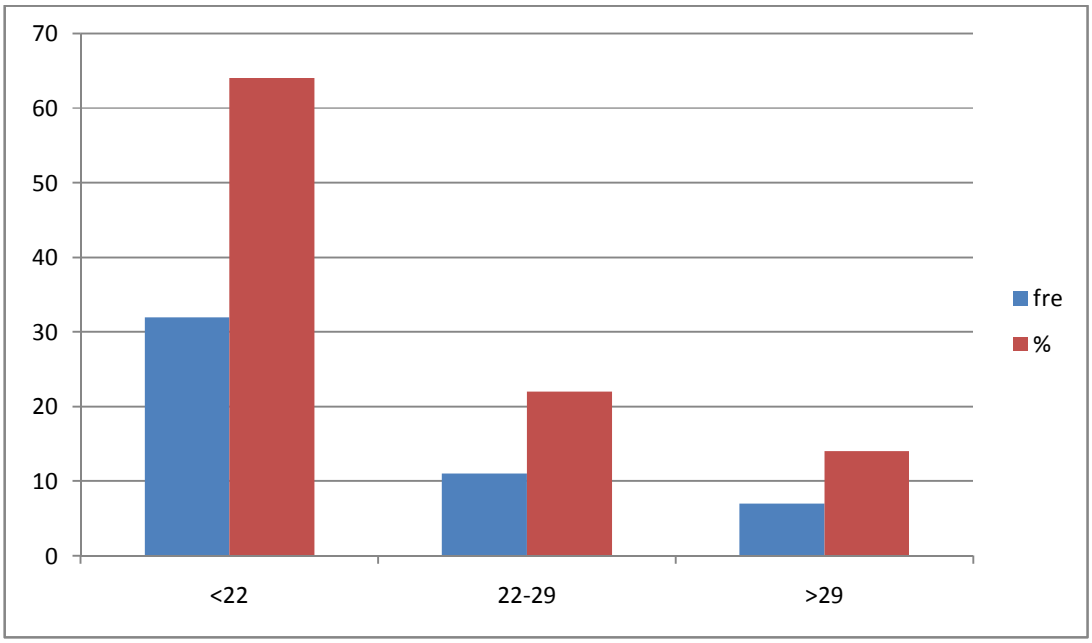
2. Duration between symptom and admitted to the hospital

Determine the peritonitis develop or not.

DIFFUSE PERITONITIES	FREQUENCY	PERCENTAGE %
PRESENT	33	66%
ABSENT	17	34%
TOTAL	50	100%

SCORING SYSTEM EVALUATION





Score	Frequency	Percentage %
<22	32	64
22-29	11	22
>29	7	14
Total	50	100

64% of the study population was in the low risk group (scores <22)

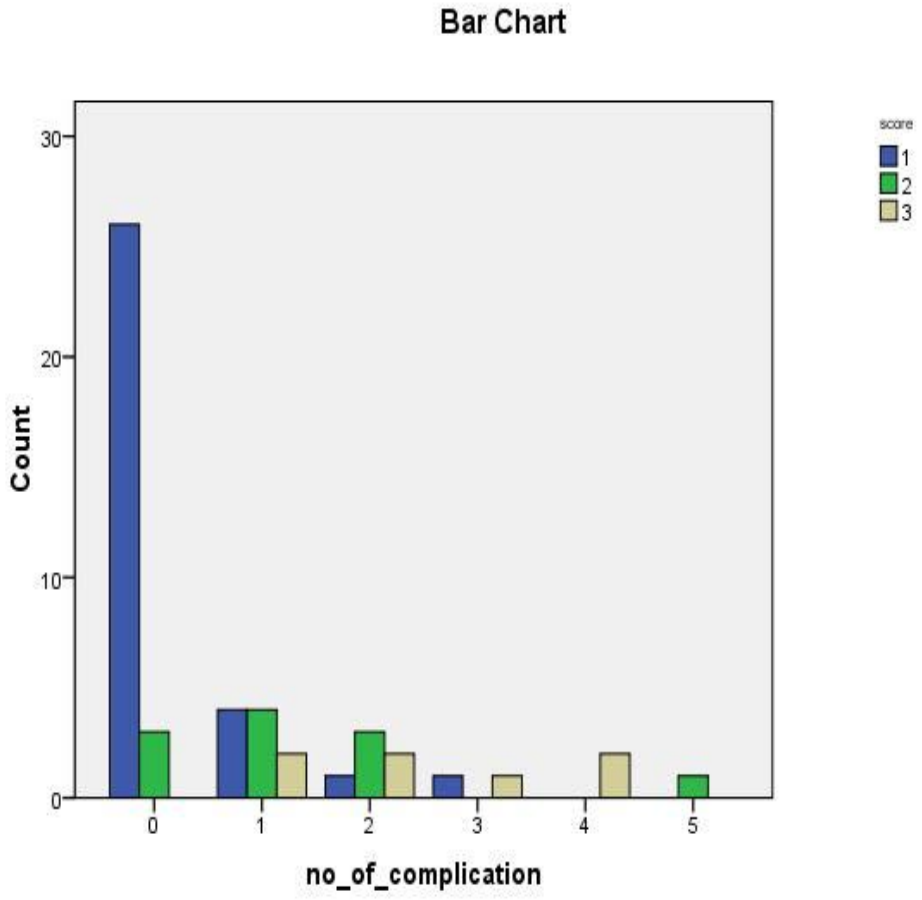
14% were in high risk (scores >29).

22% were in intermediate group (22-29).

Patients with organ failure on admission, longer duration of illness before the surgery, diffuse peritonitis, feculent exudates were more likely to have higher scores and hence fall into high risk group than their counterparts.

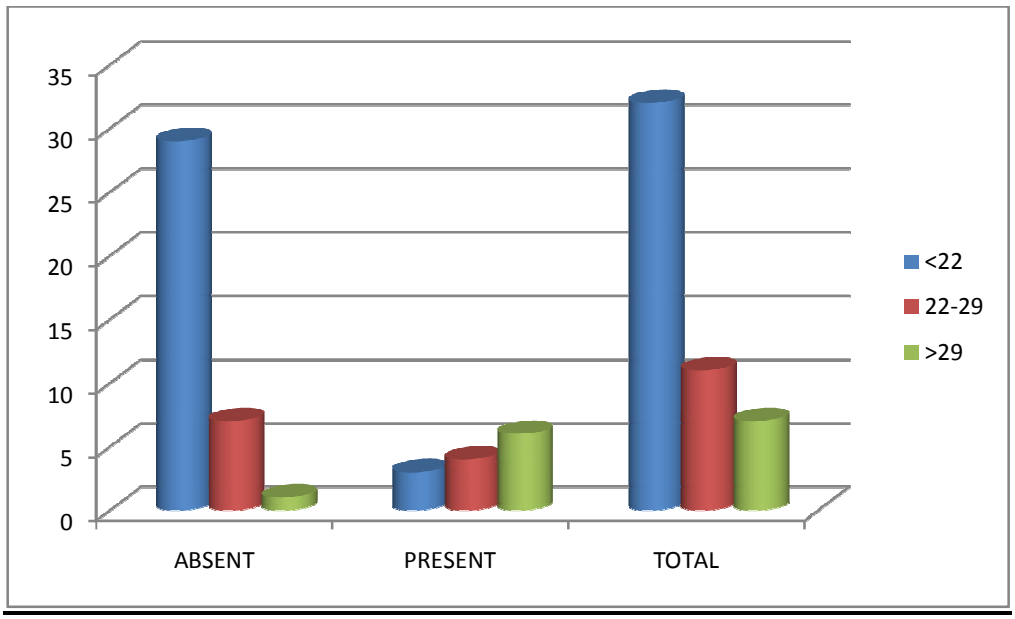
POST OP COMPLICATION

90% of the population which had no post procedure complications had a score of <22 ($p<0.002$) Whereas 70% of the patients with Mannheim peritonitis index of>29 had 2 or more complications during the post op period.



PULMONARY COMPLICATION

	<22	22-29	>29	TOTAL
NO	29 (78.4%)	7(18.9%)	1(2.7%)	37(100%)
YES	3 (23.1%)	4(30.8%)	6(46.2)	13
TOTAL	32(64%)	11(22%)	7(14%)	50



The pulmonary complications in the form of post op pneumonia, pleural effusion which required continuous monitoring of oxygen saturation, nebulisation and hence lead to longer post op recovery were significantly higher as the score increased. >90% of patients with >29 had some form of pulmonary complications which was only about< 10% in patients with score<22 ($p<0.005$)

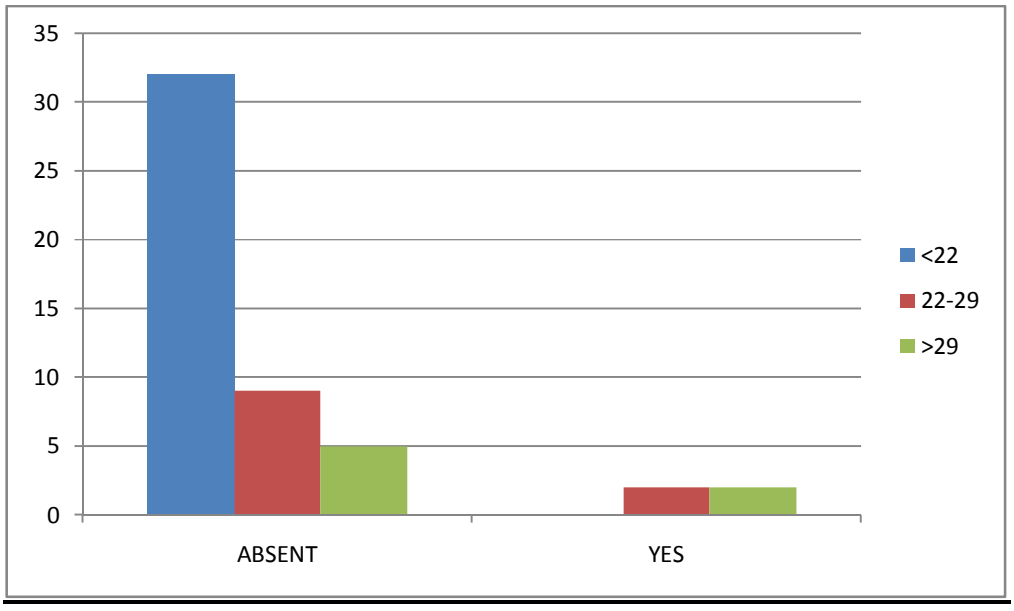
DEVELOPMENT OF ARDS

The development of ARDS in these patients was well predicted with this scoring system.

50% of the patients who developed ARDS in their post-operative course had a score of more than 29.

30%of the patient s with score >29 eventually developed ARDS in the post-operative course

ARDS	<22	22-29	>29	TOTAL
NO	32(69.6%)	9(19.6%)	5(10.9%)	4(100%)
YES	0	2(50%)	2(50%)	4(100%)
TOTAL	32(64%)	11(22%)	7(14%)	50(100%)



WOUND COMPLICATION

COMPLICATION	<22	22-29	>29	TOTAL
NO	28(75.7%)	6(16.2%)	3(8.1%)	37(100%)
YES	4(30.8%)	5(38.5%)	4(30.8%)	13(100%)
TOTAL	32(64%)	11(22%)	7(14%)	50(100%)

Up to 60% of the patients with scores > 29 developed wound related complications in the post op period which was about 40% in patients with score 22-29 and about 12% in patients with scores <22($p < 0.005$).

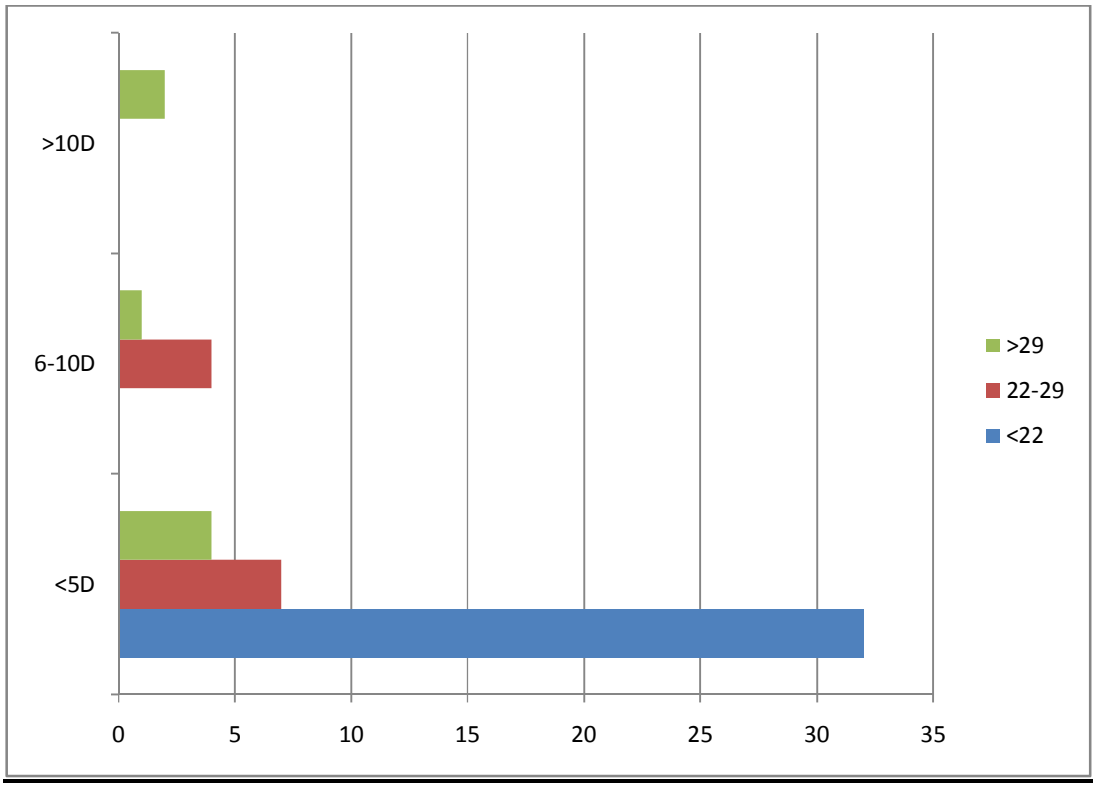
The post op complications were significantly higher in the group with score>29.

This included the surgical site infections, pulmonary, renal complications and development of multi organ failure.

There was no death in this study.

EFFECTS OF DURATION OF ILLNESS--NEED OF ICU CARE

DURATION	<22	22-29	>29
<5D	32	7	4
6-10D	0	4	1
>10D	0	0	2



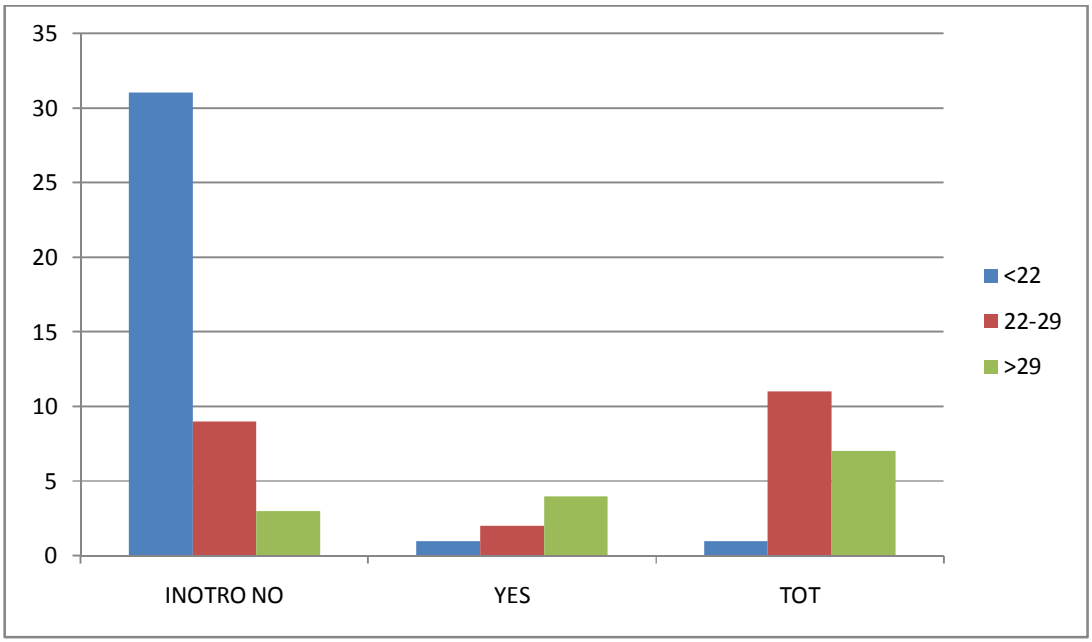
ICU stay and ward stay is significantly prolonged in patients with higher scores .There is a proportionate increase in the duration of stay with increase in scores.90% of the patients who were discharged within 10 days had a score of <22.

SCORING SYSTEM EFFECT ON SEVEITY OF ILLNESS

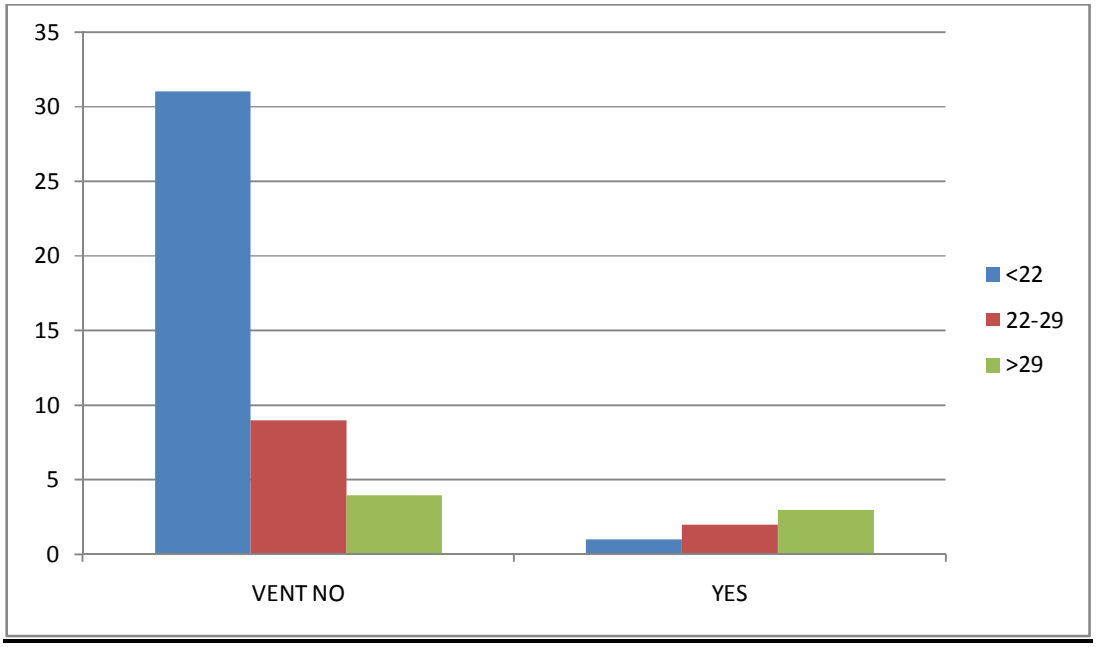
INOTROPICS SUPPORT NEEDED

INOTROPIC NEED	<22	22-29	>29	TOTAL PERCENTAGE
NO	31(72.1%)	9(20.9%)	3(7%)	43(100%)
YES	1(14.3%)	2(28.6%)	4(57.1%)	7(100%)
TOTAL	32(64%)	11(22%)	7(14%)	50(100%)

57% of the patients who required inotropic support in the post op period had a score of >29 and only 1 patient (14%) required inotropes with a score <22.



MECHANICAL VENTILATOR NEED



VENTILATOR NEEDED	<22	22-29	>29	TOTAL
NO	31(72.1%)	9(20.9%)	4(9.1%)	44(100%)
YES	1(14.3%)	2(28.6%)	3(50%)	6(100%)
TOTAL	32(64%)	11(22%)	7(14%)	50(100%)

50% of the patients who required mechanical ventilation had score of >29.

Score of >29 indicate a higher risk of need for inotropes and mechanical ventilation and need for intensive care.

OTHER INDIVIDUAL PARAMETERS

SITE OF PERFORATION& NUMBER OF COMPLICATION

Duodenal perforations >80% cases had an uneventful recovery as indicated by lesser post op complications, comparatively less requirement of inotropes and mechanical ventilation and lesser hospital stay. Ileal and appendicular perforations had higher rates of post op complications.

NO OF COMP	GAS	DUO	ILL	APP	GB	TOT
0	1	24	2	1	1	29
1	2	3	2	2	1	10
2	0	1	3	2	0	6
3	1	1	0	0	0	2
4	0	0	2	0	0	2
5	1	0	0	0	0	1
	5(10%)	29(58%)	9(18%)	5(10%)	2(4%)	50

EXUDATE

Presence of feculent or purulent exudates was reflected in higher eventual scores. Feculent and purulent exudates were associated with significantly increased post op complications requiring increased hospital stay. Up to 80% of the patients with clear exudates had no post op complications which dropped to only 30% ($p<0.005$) with the other type. However there was no statistically significant difference between feculent and purulent exudates, both having similar complication profiles.

SCORE	CLEAR	PURULENT	FECULUNT	TOTAL
<22	24(75%)	5(15.6%)	3(9.4%)	32(100%)
22-29	1(9.1%)	6(54.5%)	4(36.4%)	11(100%)
>29	0	2(28.6%)	5(71.4%)	7(100%)
	25(50%)	13(26%)	12(24%)	50

DURATION OF PAIN& NO OF COMPLICATION

COMP	<1D	1-5D	>5D
0	20	9	0
1	0	8	2
2	0	4	2
3	0	2	0
4	0	0	2
5	0	1	0
TOT	20	24	6

The study population which presented within 24 hrs of the pain onset had significantly ($p<0.05$) better outcome compared with their counterparts. This was reflected in lesser post op complications shorter ICU and hospital stay. None of the patients in this group required post op inotropes or mechanical ventilator.

As the duration of pain increased morbidity associated also proportionately increased.

ORGAN FAILURE ON ADMISSION

Patient presenting with any organ failure due to hollow viscous perforation was significantly associated with ($p<0.005$) increased morbidity.

66% of the patients with no organ failure on admission had uneventful recovery, 97% of the same population had <2 post op complications.

On the other hand 66% of the patients with organ failure on admission had >2 complications in the post op. >90% of the patients with organ failure needed post op inotropes and mechanical ventilation whereas it was the same percentage of patients who did not require inotropes in the other group. >50% of the patients with organ failure had pulmonary complications which was <3 % in the other group.

DISCUSSION

There is no ideal scoring system for the pre-operative assessment of patients needing emergency surgery.

Some pre-operative scoring systems provide approximate estimates of mortality risk but none have been shown to be sufficiently specific for use on individual patients.

At present, the Fitness Score has greatest specificity (80%) but would not be easy to use on all emergency admissions due to significantly large number (26) of variables to be collected and few variables like diagnosis of malignancy may not be available in the pre op settings.

Post-operative scoring systems such as P-POSSUM probably provide more accurate predictions, but are not useful in pre-operative assessment.

Unfortunately, there are very few studies that have revisited old scoring systems or attempted to compare systems to assess which is best.

Most articles in this field have proposed another new system.

The timing of data collection to create risk scores is seldom mentioned in the literature.

Not only do physiological values vary during the acute admission, making the scores obtained by them unreliable, but there is evidence that to include operative findings and post-operative parameters on ICU improves the accuracy of the prediction.

Although a score at initial assessment would help triage and plan treatment, comparative audit with postoperative scores remains the most useful function of scoring systems at present.

Even if accurate pre-operative predictions of outcome were possible by estimation of a risk score, an expert surgical opinion would be required to interpret these predictions at the bedside.

An experienced clinician can not only assess prognosis but also weigh up the local facilities available, the patient's quality of life and ethical issues, as well as considering the patient or relative's wishes. Scoring will never replace clinical judgment.

Scoring systems are generated and validated on specific populations that may be substantially different from the patients being scored in a different hospital. One potential resolution would be for each hospital to create a system specific to its own population, which is regularly revalidated.

This study done in Govt Stanley medical college hospital included 50 patients who presented to the emergency department and were diagnosed with hollow viscous perforation. All the patients were appropriately assessed and managed according to standard guidelines.

Few of the other studies confirmed age as a decisive factor related to mortality however this study does not show any statistical significance[30]. In other studies, patients with generalized peritonitis range from 30–66%; in our study, generalized peritonitis was present in about 66% of the patients[30, 31].

The influence of gender on prognosis has been shown of little importance in this study. Gender composition cited in other publications showed percentages, varying from 43 to 52% females and 48 to 57% male[30,31] ,72% were male in this study.

Mean MPI score reported in literature for localized peritonitis is 19 (range 0 to 35) and in generalized peritonitis, 26 to 27 points (range 11 to 43)[32, 33] which is similar to the values noted in this study.

Duration of pain >24 hours, organ failure on admission & feculent exudate were found to be independently significant factors in predicting the morbidity among the study population.

However presence of diffuse peritonitis wasn‘t a significant factor in contrast to various other studies[29]

CONCLUSION

There have been several attempts at creating a scoring system to predict mortality and morbidity risk after emergency surgery.

Some scoring systems provide a prediction that approximates to the observed mortality rate for a cohort, but none is sufficiently accurate to rely upon when considering an individual patient.

This is a validation study of the MANNHEIM PERITONITIS INDEX scoring system for predicting the morbidity and mortality in patients with peritonitis due to hollow viscous perforation.

The results of this study proves that MPI scoring system is a simple and effective tool for assessing this group of patients, and can be used as a guiding tool to decide on the management of the patient after the definitive procedure is done.

Among the various variables of the scoring system duration of pain, organ failure on presentation and presence of feculent exudates had a significant hand in predicting the eventual outcome of the patient.